105

GUY'S HOSPITAL REPORTS.

EDITED BY

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AND

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Sep. 3: Vol: 36 (1894)

VOL. LI,

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The Medical Chronicle (care of Dr. Williamson, 294, Oxford Road, Manchester).
Sheffield Medical Journal (care of Simeon Snell, Esq., 17, Eyre Street, Sheffield).
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The Pharmaceutical Journal.
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Library of Surgeon-General's Office, U.S. Army, Washington, D.C. (per Mr. B. F. Stevens, U.S. Government Despatch Agency, 4, Trafalgar Square, London, W.C.)
The American Journal of the Medical Sciences (care of Messrs. Lee Bros. and Co., Philadelphia, U.S.A.).
The Journal of Nervous and Mental Diseases (care of Dr. C. H. Brown, 25, West 45th Street, New York).
Transactions of the Odontological Society (care of the Hon. Sec., Odontological Society, 40, Leicester Square, W.C.).
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The Albany Medical Annals (per Messrs. Burdick and Taylor, 481, Broadway, Albany), care of Smithsonian Institute, Washington, D.C., U.S.A. [* Parcel to be labelled "free of duty."]
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Verhandlungen der Berliner medicinischen Gesellschaft (care of Herr B. Fränkel, Bibliothek der Berliner medicinischen Gesellschaft, Ziegelstrasse 10, Berlin. N.).
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Il Progresso Medico, Largo Pignasecca 7, Naples.
Il Policlinico, Turin (care of Professor Dr. Carlo Forlanini, Turin).

SEQUEL TO THE CASE

OF

FRACTURED PELVIS WITH RUPTURED URETHRA,

Which was published in the 'Guy's Hospital Reports,' Vol. XLIV.

By L. A. DUNN, M.S.

The chief points reported in the above volume are as follows:—Mr. D—, æt. 40, was thrown from his horse and crushed by the animal on June 9th, 1884. He sustained a fracture of the pelvis on both sides of the symphysis pubis, which was displaced backwards, with complete rupture of the urethra and considerable damage to the soft parts of the right thigh. An incision was made into the perinæum, but all attempts to find the proximal end of the urethra failed.

The urine escaped for the most part through the perinæal opening, but some extravasated down the right thigh and caused an abscess to form in the popliteal space. After this had been opened urine flowed freely from the drainage-tube which had been inserted here, and, indeed, about one third of the whole urine passed escaped by this popliteal opening.

On July 8th an anæsthetic was again administered, but

all attempts to reach the bladder from the perinæum failed. The operation had the effect of making free the passage through the perinæum, and thus allowed the other sinuses to heal.

The patient progressed favorably, and was able to walk three miles by the middle of December. He passed the next year, 1885, without any serious trouble, but had an attack of retention of urine early in 1886, which was relieved by means of aspiration above the symphysis pubis.

In March, 1887, the report states that there was a tiny opening in the perinæum into which a silver probe could be introduced, but only to the distance of three quarters of an inch. Through this opening the whole of the urine, amounting to between three and four pints daily, escaped freely. A specimen was examined and found to be acid, with a sp. gr. of 1025, and it contained a little pus and albumen, not more of the latter than might be accounted for by the pus. His general health was good, and he refused to have any further operation performed, stating that he would "let well alone."

He amused himself with shooting and other country sports, and really felt very little inconvenience resulting from the accident till February, 1889, when his health began to fail. He suffered some pain when he passed his water, the whole of which was now but slowly voided through the perinæal fistula. The temperature became irregular, and his face pallid and anxious. At times he would remain in bed for the day, and lost interest in outdoor sports. It was about this time that a thick sediment, which varied in quantity from day to day, was noticed in the urine. This sediment consisted of pus cells, but except for this and the presence of a little albumen, which was no doubt due to the pus, the urine was normal.

There was some ill-defined resistance in the left iliac region, and considerable induration on the left side of the bladder and prostate, palpable from the rectum. He was watched for another two or three weeks, and, as he seemed to be rapidly taking a downward course, it was decided to do an exploratory operation, and, if possible, evacuate the pus which had doubtless collected in the pelvis. There was by this time, March 30th, 1889, a considerable amount of brawni-

ness in the left inguinal region and induration in the pelvis, which could be grasped bimanually, one finger in the rectum and the fingers of the other hand externally. Under chloroform an incision was made just above and parallel to Poupart's ligament on the left side, and, after cutting through a considerable depth of inflamed tissue, pus was reached with a director, and the abscess was opened up by means of dressing forceps. About half a pint of pus escaped, and the cavity, after being well flushed out, was drained and dressed.

The patient improved rapidly, so that by the 14th of April he was able to be brought up to London, and was placed in Bright Ward under the care of Mr. Durham.

A consultation was held, and it was decided to attempt to reach the bladder from the perinæum. Under chloroform it was found possible to introduce a fine director only to a distance of an inch and a half up the fistula, so contracted and devious had this become; hence Mr. Durham, with his index finger on the apex of the prostate, incised the perinæum as in Mr. Cock's method. After considerable difficulty a Brodie's director was passed into the bladder, and a No. 12 catheter introduced along it and tied in.

The patient made a rapid recovery, and left the hospital on May 9th, having been taught to pass a catheter into the bladder from the perinæum. No attempt was made to re-establish the continuity of the urethra, since it was thought that the patient would be able to keep a short straight passage open better than if it were a long devious one.

On returning home the patient resumed his active life, and suffered no trouble whatever with his urinary apparatus. He passed a No. 12 catheter regularly every third or fourth day, and continued to do so for several months. He gradually became less regular as regards the use of the instrument, and as No. 12 was somewhat difficult now to introduce, and caused him a little pain, a smaller one was procured, and so on until it was only with the greatest difficulty that a No. 6 catheter could be passed into the bladder.

He was advised to return to Bright Ward and have the fistula dilated. This he accordingly did on April 19th, 1893, and Mr. Durham again operated. A director was passed

into the bladder, and the walls of the fistula were divided by means of a straight bistoury in the direction of the four points of the compass.

A large tube was inserted, and the bladder thus drained. A slight attack of cystitis resulted, but with this exception the recovery was uninterrupted, and the patient left the hospital on May 2nd.

In order to prevent the channel contracting again, a solid, flexible india-rubber stem, the size of a No. 12 catheter, and about four inches long, projecting from a small india-rubber cushion, was made by Messrs. Down, Bros. To the cushion four webbing straps were attached, which could be fixed to a waist-band. The stem was introduced into the fistula, and then kept in position by fixing the straps to the waist-band. Instructions were given to the patient to oil the stem and wear it every night, and all night, leaving it out when he arose in the morning.

Upon inquiry as to how the stem acted, he wrote as follows on December 13th, 1894:—"I have had five plugs in all, but two are still very good; the three were ruined by the use of oil and vaseline; since I have used glycerine they have stood their work well. I use one almost every night, and only a No. 12 catheter occasionally, but I always find the latter goes into the bladder quite easily."

The stem does not produce any leakage from the bladder, as its extremity does not quite enter that viscus. The urine is voided only three or four times daily, and flows almost entirely through the perinæal opening. In answer to a question on this point, he remarks in the above letter, "After the first operation at Guy's, I was able to pass quite half the water naturally, but since the second operation the natural passage has been almost stopped, not more than a few drops passing that way." He then continues, "I suppose I still now and then have stricture, that is, the present passage becomes contracted for a time, but I have not yet, since the last operation, had to use the catheter to get rid of the water. I fancy I sometimes have a bit of a pelvic abscess, I feel weight and pain at the bottom of the stomach, and I get a bit feverish; then there is a discharge from the fistula for a day or two, and all goes right again. I am remarkably well altogether,

and can stand a long day's shooting without getting at all tired."

It is now a few months more than ten years since the accident, and I think we may fairly hope that the natural tenure of life will not be shortened by this most severe injury.

TEMPERATURE AFTER OPERATIONS.

Abstract of an unpublished Address on Surgical Fever, made shortly after its delivery in the year 1879.

By THOMAS BRYANT, M.Ch.

The author based his remarks on "surgical fever after operations" on an analysis of ninety consecutive cases of great operations which had occurred in his practice at Guy's Hospital during the years 1877–8. His object was to show that the immunity from such fever, which the advocates of the spray system claimed so persistently to belong to their practice, and to theirs alone, was shared with others; and that the antiseptic methods which he adopted, and which he believed other surgeons adopted, were not in the majority of cases submitted to operation followed by any persistent elevation of temperature, or even by any elevation at all after the first day or so; and also that the constitutional disturbance associated with the rise in the temperature was rarely severe. With the view of demonstrating these points he divided his cases into three classes:

The *first* including thirty-three operations for tumours of soft parts, as of breasts, &c.

The *second*, thirty-seven operations on bones and joints.

The *third*, twenty operations involving cavities, such as the abdominal or pelvic.

All these cases were tabulated and numbered, and the

highest temperature registered during each day for about a fortnight after the operation quoted. He expressed a regret that morning and evening temperatures had not been in all the cases so systematically taken at the same hours as he would have wished, but in spite of such default he thought the object he had in view would be achieved; the material at any rate being highly suggestive of truth, if not scientifically conclusive.

In the *first* class of cases, that of tumours, there were 33 cases, 20 of which were of the breast; and all were successful.

In eleven, or a third of the whole number, the temperature during the progress of the case never rose above normal. In thirteen it rose to 100° or 102° on the first day after the operation, to fall in ten of the cases to the normal point during the next two days, and in the remaining three within the week. In five cases the temperature rose to 100° or more on the second day, when on the first it had not been high, but in all to sink within a day or so to the natural standard; and in four cases it rose on the third day to the higher point, but to decline in two or three days.

In only two cases out of the whole number was the temperature of 100° maintained for more than two or three days.

In the *second* class—including operations on bones, amputations and excisions—there were 37 cases, three of which were fatal: one from gangrene, another from broncho-pneumonia, and the third from diphtheria. In each of these fatal cases the temperature rose within three days of the operation, and never fell.

Of the 34 cases which recovered, in ten the temperature never reached 100° or above normal. In thirteen it reached 100° to 102° on the first day, but in all to fall on the second, and not to rise again. In eight the temperature rose to 100° on the second day, when it was low on the first, and in all subsequently to decline; and in three cases the temperature rose on the third, fifth, and seventh days respectively, but in all to fall. In some five or six of the cases the temperature rose again during their progress for one or two days, but never to last; the temporary elevation being usually easily

explained by some bowel condition, or retention of fluid in the wound.

The *third* class of cases included operations on the abdominal and pelvic-cavities, and numbered 20 cases, two of which died, one from kidney disease, and the other after gastrostomy. In nine of these the temperature never rose, and in four it rose on the second, fourth, fifth, and sixth days respectively, but only for the day. In seven it rose on the first day to 100° or 102°, to fall in six of the cases to normal during the next two days, and only to rise again in three on one or two occasions.

In analysing the 90 cases it was shown that in thirty, or one third of the whole number, the temperature never rose to fever point, or up to 100°. In thirty-three it rose on the first day to 100° or 102°, to fall during the next two days. In fourteen it rose on the second day to 100° when it had been low on the first; and of the remaining ten cases the temperature rose only for a day during the first week. In all the cases the method of dealing with the wound during the operation, and of dressing the wound subsequently was alike.

In the majority of cases the arteries were twisted, in exceptional instances ligatured with carbolised catgut. In all, the wound after the operation was washed with warm water with every pint of which about two drachms of the tincture of iodine had been mixed, making it of a light sherry colour.

In all, the sponges that had been used were washed in a similar solution, and in most a hot sponge was applied to the surface of the wound before it was closed, to check capillary bleeding. In most of the cases the edges of the wound were brought together by means of silk or wire sutures alternating with some narrow strips of strapping, and provision was invariably carefully made for effective drainage, with or without drainage-tubes.

In all the cases the surface of the wound was covered with lint saturated with a solution of terebene and olive oil (1 part to 5), and in most a pad of sponge, lint, cotton-wool, or carbolic gauze was applied with some amount of pressure. Where limbs or joints were involved the application of a splint was universal.

In the majority of cases the wound was not dressed till the third day, and then only when there was the slightest suspicion of any retained secretion as indicated by local pain or some elevation of temperature. In dressing the wounds also the greatest care was observed not to disturb the parts more than was absolutely necessary.

In the year 1879 the author stated that he had not had one example of pyæmia.

From these facts it was concluded that in one third of the cases of the author's operations at Guy's Hospital there was no surgical fever after operation; in another third it appeared on the day following the operation, to disappear on the second or third day; in one sixth it appeared on the second day, to disappear on the third or fourth; and in the small remainder the temperature rose for a single day during the first week.

When the temperature remained high for many days consecutively after an operation the author stated that the prognosis was always bad.

In conclusion the author drew attention to the statistics of Volkmann on surgical fever as given in 1878 ('Med. Times and Gaz.,' June 22nd, 1878), in which it was shown that, after severe operations performed with the true Listerian precautions of the day, in one third of the cases there was no fever; in one third there was moderate fever; and in one third high fever. He also pointed out how his own statistics and proportions were in the rough analogous to Volkmann's, except that in his own there was a smaller proportion of cases in which high fever existed.

He reminded his hearers that Volkmann called the fever in his cases "aseptic," but he presumed that he had the same right to use a like term, since in his own cases putrid elements were carefully excluded or guarded against. He said, moreover, that in his cases the results were brought about by such simple means as were open to every practitioner in this country.

The paper of which the above is an abstract was never published, but it clearly shows what Mr. Bryant's practice was in 1877–8; and now, seventeen years later, we have reason to believe that no serious change has to be recorded,

for at the present time Mr. Bryant tells us that he still employs iodine water to clean his wounds, and is as careful as ever to apply sponges wrung out of hot iodine water to check capillary oozing before he brings the edges of his wounds together. He now dusts all fresh wounds, before he closes them, with a powder composed of one part iodol and four parts of boracic acid. To the surface of the wound he applies a dry dressing, such as a few layers of iodoform gauze, well covered in with one or two layers of wood-wool tissue, or the wood-wool tissue alone.

For deep wounds he always introduces a drainage-tube at the most dependent angle of the wound, but he never passes the tube through the wound as if it were a seton. He rarely leaves the drainage-tube in the wound beyond the second or third day. He always employs torsion to his arteries, and uses carbolised or chromicised catgut to such veins as may require attention. On the second or third day after the operation the bandage is so cut as to allow of the drainage-tube being removed or looked to, but the wound is rarely fully dressed till the third, fourth, or fifth day, the indications for dressing being the freedom of secretion, pain, or elevation of temperature. As a rule, Mr. Bryant expects to secure quick union in most of his cases, and generally obtains it.

It may be added that Mr. Bryant uses less opium after operations than he did, in the belief that opium in all its forms checks repair ; when employed he uses it generally in the form of a suppository.

ON EMPYEMA FOLLOWING LOBAR PNEUMONIA.

By W. HALE WHITE, M.D., and A. CHANING PEARCE, M.B.

The subject of empyema as a complication of lobar pneumonia is so scantily treated in most text-books, that we thought it would be of interest to publish the following twenty-six cases which have occurred at Guy's Hospital from 1884 to 1894, both years inclusive. Probably in that time more than twenty-six cases have been seen in the hospital, but we have been very rigorous in excluding those in which there was for any reason the slightest likelihood that the patient was suffering from broncho-pneumonia rather than lobar pneumonia. Nor have we included a case under one of us in 1893 in which the constitutional signs of pneumonia were present, but the pneumococcus, having invaded the pleura, had produced an empyema without any accompanying pneumonia. It was recorded by Dr. Washbourn,[1] and perhaps some of our twenty-six cases resemble it; but as attention has only been recently directed to the question it is impossible, from reading the report of one or two of the older cases, to decide this, and therefore they have been included here as they are indexed as cases of pneumonia. It is clear that it is impossible to state quite accurately the proportion of cases of lobar pneumonia which are complicated by empyema, because, as just mentioned, we may have excluded some cases of lobar pneumonia which resembled broncho-pneumonia.

The relative frequency with which during recent years empyema has complicated pneumonia may be seen from the following table.

[1] 'Med.-Chir. Trans.,' 1894.

TABLE I.

	1894.	1893.	1892.	1891.	1890.	1889.	1888.	1887.	1886.	1885.	1884.	1883.
Total cases of lobar pneumonia	67	182	88	108	88	60	43	72	48	39	26	69
Number complicated by empyema	2	9	2	6	1	1	1	2	0	0	1	1
Percentage complicated by empyema	3·0	4·94	2·27	5·5	1·13	1·6	2·3	2·9	0	0	3·8	1·4

That empyema has been a more common complication since 1890 than during the decade from 1880 to 1890 is shown not only by the above table, but also by the fact that during that decade Messrs. Hadden, Hector Mackenzie, and W. W. Ord[1] found empyema in six out of 708 cases of pneumonia during the years 1880–90 at St. Thomas's, or in 0·84 per cent.; but the above shows that during 1891–4 empyema occurred in 4·27 per cent., or in five times as great a proportion of cases as during the years 1880–90 at St. Thomas's, and in two and a half times as great a proportion of cases as during the years 1883–90 (both inclusive) at Guy's.

Mortality.—One of the twenty-six cases left the hospital of his own accord because an operation was suggested. Of the remaining twenty-five nine died, a striking contrast to the fact that of the six St. Thomas's Hospital cases of pneumonic empyema none died; and this is not accounted for by the circumstance that during the years 1883–90 empyema was an unimportant complication, for three out of the seven cases in Guy's during those years died. Out of nine cases of empyema in 1893 four died, but the general mortality from pneumonia at Guy's that year was very high, and we know that the mortality from pneumonia is very varied: thus at St. Thomas's in 1887 it was 35 per cent.; in the latter half of 1888 and the first nine months of 1889 it was 9 per cent.; in 1882, 7 per cent.; but in January and February, 1890, when influenza was rife, it was 64 per cent. There is nothing noteworthy about the sex and age of the fatal cases.

[1] 'St. Thomas's Hosp. Rep.,' vol. xix.

Two of our cases should be separated from the rest, as they developed malignant endocarditis, which, as is well known, occasionally complicates pneumonia, for the pneumococcus can thrive on the valves of the heart. These cases were Nos. 6 and 25. Neither patient gave a history of having had rheumatic fever, but both a brother and a sister of the second had heart disease. Neither had any cardiac murmurs on admission, nor during the time they were under observation. The first patient was under our own care. He for the first few days had a rub where the pus was found; this disappeared when the temperature fell, but returned again. The temperature dropped gradually, and reached normal on the fourteenth day. There was no crisis. It began to mount three days later, but did not become at all high. During this second period of pyrexia he seemed very ill, and had much diarrhœa. We did not explore his chest earlier because we did not think he had much fluid, and in this we were correct, for the empyema only contained five or six ounces. It was interesting to notice that the lung contained several abscesses in the neighbourhood of the empyema, and that the malignant endocarditis was more marked on the right side than the left, as though infection had taken place from the pleura through the azygos veins. In the other case the temperature fell to normal on the fourth day, and began to rise again on the tenth. The empyema was found and evacuated, but the patient ultimately died. The infection presumably took place through the pulmonary veins, for only the left side of the heart was affected, and there were several abscesses in the spleen. Both cases went downhill rapidly towards the end.

The nineteenth case is closely allied to these, although there was no actual endocarditis; but the patient had an aneurysm of each branch of the hepatic artery, and these were probably, considering the patient's age, infective, some coagulum containing pneumococci being perhaps carried from the recesses of the left ventricle. This patient's temperature fell to normal on the eleventh day, and it came down gradually and rose again and assumed the hectic type on the fourteenth day.

To sum up, we may say that general infection occurred in three out of twenty-five cases of pneumonic empyema, or in

12 per cent. of the cases; and that although there is after the period of pyrexia which is coincident with the pneumonia an apyrexial period of a few days before the onset of the secondary period of pyrexia, which continues till death, yet in two at least of our cases it is noteworthy that the primary stage of pyrexia terminated by lysis. We find that at St. Thomas's one case of malignant endocarditis occurred in the series of 708 cases of pneumonia, but at Guy's we had two or three among 890 examples of pneumonia.

We will now consider the cases in which there was no evidence of general infection. In them the diagnosis of the presence of pus may be difficult or easy. It is, of course, very important to pay most careful attention to the physical signs, but we do not think any useful result would be achieved by discussing them by the light of the cases we have collected here, for it is not always possible to satisfactorily compare descriptions of physical signs by different observers. Our own experience is that in empyema after pneumonia—more than is the case with other collections of fluid in the chest—it is particularly easy to make mistakes as to the amount, probably because the extensive consolidation of the lung modifies the signs of fluid. Among the general signs indicating the presence of pus, much loss of flesh and anæmia are not very common,—partly if not entirely, no doubt, because the pus is usually evacuated soon after it has collected. In many cases the pain during the attack of pneumonia is very severe, and profuse sweating and diarrhœa, both before and after the pneumonic crisis, are often observed; but whether any of these symptoms are more frequently present in those cases in which an empyema follows pneumonia than in uncomplicated pneumonia we are not prepared to say. We, however, attach much importance to a local tender spot as indicative of pus, and if œdema of the chest wall is present it is a valuable aid to diagnosis. The temperature, respiration, and pulse each deserve a separate consideration.

We have looked through each of these cases in which there was no evidence of general infection, and four of them, viz. Nos. 3, 5, 23, and 24, died so early in the disease that the temperature could be no guide to the presence of pus,

Table II.

No. of case.	Day of disease crisis occurred.	Day of disease second rise of temp. began.	Day of disease pus was evacuated.	Amount obtained in fl. oz.	Remarks.
1	9	12	18	40	
2	8	11	25	9	The 9 fl. oz. were aspirated. Temperature remained down for a week after, and then began to rise again, so on 37th day chest laid open, and 10 fl. oz. escaped. From that did well.
3	11	—	13	20	Death. Second day 3 fl. oz. pus aspirated from left chest.
4	9	13	33	12 in chest, 23 in pericardium	Death.
5	—	—	6	16 in right chest, small empyema in left	Death before temperature fell.
6	8	12	21	5½	Death. Malignant endocarditis.
7	10	12	18	20	
8	8	16	40	½	
9	22	42	49	4	Pericarditis.
10	8	11	18	80	
11	10	13	20	7	
12	7	9	—	—	Patient went home.
13	5	8	17	72	
14	—	—	25	7	Death. Pericarditis.
15	12	15	42	Little	
16	9 (?)	10	28	12	
17	7 (?)	12	45	15	
18	10	—	25	Small	Ninth day 8 oz. of fluid aspirated.
19	11	14	24	2	Malignant hepatic aneurysms. Death 34th day. Second empyema right side.
20	No	Fall	16	20	Twelfth day 4 oz. of fluid aspirated.
21	7	8	24	9	
22	5	6	18	8	
23	—	—	3	13	Death.
24	—	—	4	8	Death. 5 oz. of pus aspirated a few hours before death; the other 3 were found at the autopsy.
25	8	16	26	170	Death. Malignant endocarditis.
26	11	16	20	22	Twentieth day 20 oz. of pus aspirated; on 26th 2 oz.; on 27th chest opened.

and the report of the temperature in Nos. 14 and 19 is not full enough for us to draw any deductions, and in Case 12 the empyema probably burst through the lung. We have attempted to group the remaining cases into two classes, one in which there was a distinct apyrexial interval between the crisis of the pneumonia and the secondary rise which indicates the probability that pus is present, and another in which no apyrexial interval is seen. It is impossible to draw an absolutely rigid line between the two groups, for there are cases in which the temperature having been previously very high falls to about 100° for a day or two, and then begins to ascend again. For diagnostic purposes this might be called an apyrexial interval, although the fall never reaches normal. Still it appears to us that Cases 1, 2, 7, 9, 10, 15, 17, 22, and 26 may be fairly placed in the group characterised by an apyrexial interval. This was usually short. Thus in Case 24 it was one day, in Cases 1, 2, 7, 10, and 15 it was two or three days, and in Cases 17 and 26 four or five. Case 9 was remarkable and quite exceptional, for the temperature did not become normal for twenty-two days after the first rigor, then it was nearly normal in the morning and about 100° in the evening for three weeks, then for a week it was considerably higher and hectic. At the end of this week the pus was found and evacuated, but only four ounces were present. Possibly the peculiar temperature of this case was due to the pericarditis which complicated it.

We had put Cases 11, 13, 16, 20, and 21 into the group in which there was no apyrexial interval, but, as we have already remarked, there is no distinct boundary line, and Cases 11, 13, and 21 might very well go into the group characterised by an apyrexial interval, for in the first two there was a period of a few days in which the temperature was lower than during either the pneumonic period or that indicative of pus, and in which it occasionally was normal in the morning although high in the evening; and in the 21st case there was a crisis with a fall to 99·4°, although the next day the temperature rose again. But in Cases 16 and 20 there was no apyrexial interval, and in the second of these the temperature remained consistently high.

It appears, therefore, that, leaving aside the malignant endocarditis cases, we may conclude that when an empyema is going to complicate a pneumonia the temperature will fall at the date at which the crisis of the pneumonia may be expected; that it will then keep somewhere between normal and 100° or a little over for a period varying from one to four days, after which it will begin to mount again. There is a rare group of cases in which the temperature keeps high—of which Case 20 is a good example,—and another rare group in which the temperature indicative of pus is very long delayed, and of this Case 9 is an instance.

If pus is present the temperature after the pneumonic crisis is such as usually indicates pus, viz. it is much higher in the evening than the morning. The charts of Cases 4, 8, 10, 11, and 13 show this very well, and it was equally true of the cases of which charts are not published. It is very important also to notice that in Case 3, in which pus was present from the beginning, although the patient undoubtedly had pneumonia the temperature was always higher—often considerably higher—in the evening than the morning, and the chart looks like the chart of a patient with pus. In other cases, too, the temperature from the very first—before the crisis, if one has taken place—has been of this character; see, for instance, the charts of Cases 4, 13, and 20. In Case 13 seventy-two ounces of pus were let out on the seventeenth day after the initial rigor; in Case 20, twenty ounces on the sixteenth day; and in Case 4, although the signs of fluid in the chest did not show themselves early in the case, yet a pericardial rub was heard on the seventh day, and at the post-mortem there were twenty-three ounces of pus in the pericardium. It appears, therefore, that at whatever period of a case of pneumonia pus begins to collect the temperature will become hectic,—a fact difficult to understand if the pneumococcus is the cause of the outpouring of pus, for it leads to neither pus nor a hectic temperature when it invades the lungs only.

In considering the pulse we must leave out of consideration Cases 6, 19, and 25, as they are instances of malignant infection, Cases 4, 9, and 14, as they had pericarditis; Cases 5, 23, and 24, as they did not live long; and Cases 7,

18, 21, and 22, as the records of pulse are not sufficiently full to be valuable. Of the remaining cases we would direct attention to Cases 1, 2, 8, 10, 11, and 12, because in all of them the pulse was slower during the pyrexia, which coming on after the crisis showed pus to be present, than when the temperature was at the same height before the crisis: for instance, in Case 2 before the crisis the temperature was usually between 102° and 103°, the pulse varied between 110 and 148; after the crisis, from November 15th to November 30th, the evening temperature was often over 102°, and the morning temperature was a degree or so lower, the pulse varied between 80 and 104, and from December 3rd to December 10th the morning temperature was about 99°, and the evening temperature was between 100° and 103°, but the pulse varied between 76 and 100. The cases are too few, and the difference in many of them is too slight, for any certain deductions to be drawn; but they suggest that sometimes invasion of the pleura by the pneumococcus does not quicken the pulse so much as invasion of the lung, and it is interesting in this connection to observe that in Case 13, in which pus was probably present from the beginning, the pulse was rather slow for the height of the temperature. If, however, the suggestion here thrown out be true for some cases, it is not for all, for no noteworthy difference in the pulse during the two periods could be observed in Cases 15, 16, 17, and 26; and although pus was present from the first in Case 3, yet the pulse was not unusually slow.

The next point to which we would direct attention is the rapidity of respiration. Cases 5, 23, and 24 died too soon, and Cases 14, 18, 21, and 22 are reported too briefly for the observations to be of much value, and we need not consider Cases 6, 19, and 25, in which general infection occurred, nor Cases 9 and 11, in which the respiration was never very rapid. Of the remainder, in the greater number, when pus was present, the rapidity of respiration was only what might have been expected, considering that there was fluid in the pleural cavity and the temperature was elevated. In a few cases the respirations appeared rapid during the purulent stage when contrasted with the antecedent pneu-

monic stage, but a special explanation was possible in some, —for example, in Case 12 the empyema burst into the lung, and this probably set up considerable bronchitis, and in Case 13 the amount of pus was very large, and in Case 20 it collected very quickly; but occasionally the cases suggested that rapidity of respiration during the purulent stage was not capable of a mechanical explanation,—for instance, the crisis occurred on the night of the tenth day in Case 3, but on the eleventh and twelfth days although the temperature was very near to 98·4°, yet the respirations remained much the same as before the crisis, when the temperature was often over 104°, and in this case it is hardly likely that the fluid increased so rapidly during the crisis as to compensate for the fall in the rapidity of respiration which might have been expected. In Cases 16 and 17 also the fluid collected so slowly that it seems hardly likely that its presence satisfactorily explains the fact that the respirations were more rapid during the secondary pyrexia than the first.

Table 2 shows that the amount of pus at death or on evacuation was most variable. The greatest amount was 170 ounces in one of the malignant endocarditis cases; but leaving out these cases of general infection, we find eighty ounces in Case 10 and seventy-two in Case 13. If in both these cases we believe that the formation of pus began with the onset of the pneumonia, at least five ounces must have formed daily; and, if we consider that the pus did not begin to collect until the secondary rise of temperature began, the rate of collection was about half a pint a day. Case 8 is very interesting, for only half an ounce was evacuated, although twenty-four days had elapsed since the secondary rise began. Between this case on the one hand, and Cases 10 and 13 on the other, there are all sorts of variations in the quantity of pus, and the rate at which it collected. The average time between the beginning of the second period of pyrexia and the evacuation of pus was 13·2 days; but as the figures from which this average was obtained vary between 4 and 33, it is of little or no value.

In Cases 2, 3, 18, 20, and 26 the chest was aspirated, but in every instance the pus collected again. In Case 3 a pint was found at death eleven days afterwards, and the aspira-

tion did not improve either the physical or the general signs. In Case 2 aspiration improved both for a time, but after a week the temperature began to rise, and the chest became duller, and had to be laid open. In Case 18 aspiration improved both for a few days, but here also the chest had ultimately to be laid open. In Case 20 hardly any benefit followed aspiration, and in Case 26 the temperature fell and the patient was better for a day or two, but then it began to rise again, and ultimately in both these cases the chest was laid open.

We are afraid that this collection of cases does not teach much that is not common knowledge, but the points that seem to be brought out are that empyema has been a more common sequel of lobar pneumonia during the last few years than formerly; that the chief evidence of its presence is that physical signs of fluid are found in the chest at a period when we should expect to find those of resolution of the lung after pneumonia; that often extreme local tenderness develops; and that after the temperature has remained nearly at normal for about two or three days after the crisis it slowly begins to rise again, and is then considerably higher in the evening than the morning. It seems clear that aspiration is futile, and when there is no doubt that pus is present the chest should be laid open and drained at once. We ought, perhaps, here to add that one of us has seen a patient in whom an empyema following pneumonia burst into the lung, and the pus was coughed up. As the temperature immediately fell and remained normal no operation was done, and the patient made an excellent recovery.

Points of less importance are that there is a small group of cases in which the empyema is associated with malignant endocarditis and general infection, and another small group in which pus is probably present from the very first, and in these the drop of temperature is wanting. There is some evidence that occasionally when an empyema follows an acute lobar pneumonia the pulse and respiration are rapid, out of proportion to the height of the temperature and the amount of fluid present.

APPENDIX OF CASES OF EMPYEMA FOLLOWING LOBAR PNEUMONIA.

Case 1 (1894).—Ernest A—, æt. 16, admitted under Dr. Goodhart in John Ward February 28th, discharged April 18th, 1894; clinical clerk, Mr. H. M. Stewart.

History.—Mother, and probably father, died of phthisis.

February 22nd.—Came home early from work with malaise and shivering. Since then he has had diarrhœa, and temperature about 103°. Twenty-four hours before admission he began to perspire, and he has done so ever since.

Condition on admission.—February 27th, 10 p.m.—Patient in much distress and pain; face flushed, skin hot and dry. *Signs: Right lung:* Occasional rhonchi are the only abnormal sounds. *Left lung,* in front: Note impaired down to fourth rib; this area also gives bronchophony and increased tactile fremitus. Expiratory murmur is harsh and prolonged. Below fourth rib and in axilla note is also impaired, and voice- and breath-sounds are deficient. Behind: Note very impaired up to spine of scapula; over this area voice- and breath-sounds are deficient. Distant tubular breathing near angle of scapula. There is ægophony over considerable area at back. There is deficient movement on left side. *Heart:* Impulse rather diffuse and feeble; slightly displaced to right.

Treatment.—Ice poultice during first night gave no relief, linseed poultice was much appreciated. Tinct. Opii ℥v rep. bis., Am. Carb. gr. iv in Mist. Am. Acet. 6tis horis, brandy ʒiij, milk Oij.

February 28th.—Fairly comfortable all day, restless at night, and was therefore given Tinct. Opii ℥v, and that failing, Inject. Morph. Hypoderm. gr. $\frac{1}{6}$.

March 1st.—Had good night, and is fairly comfortable this morning. Sputum rusty. *Signs:* Note impaired behind a little above spine of scapula. Ægophony occurs below this and at the base; both voice- and breath-sounds are very deficient. Difference in breathing between the two sides in front is not so well marked.

3rd.—Slept well after morphia.

4th.—Had no Inject. Morph. Hypoderm. last night; seems much better this morning, temp. 98°. *Signs:* Dulness behind not so high. Tubular breathing not so marked. Air enters much better in axilla and above fourth rib in front.

6th.—*Signs:* No râles or tubular breathing.

8th.—*Signs:* Dulness in front below third rib, with ægophony over this area. Air can be heard entering over a small area over eighth and ninth ribs.

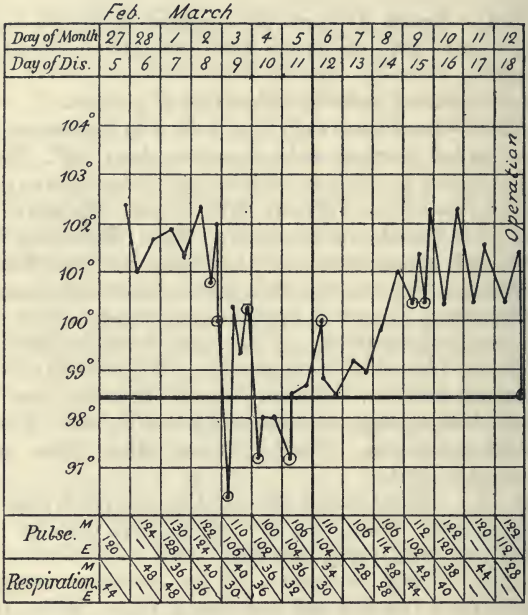
10th.—*Signs:* If anything the dulness has increased. Needle put in and pus found.

13th.—*Operation:* Pus evacuated through eighth space; it amounted to about two pints. *Signs:* Note less impaired; air can be heard entering down to tenth rib; still tubular breathing. Cardiac impulse now at left border of sternum.

20th.—*Signs* as on the 13th; entry perhaps better, and note improved.

April 2nd.—*Signs:* Air enters fairly down to ninth rib. Chest falling in; note fairly resonant. Tube taken out.

17th.—Wound healed. *Signs:* Fair resonance and fair entry, at left base.



Case 2 (1894).—Thos. F—, æt. 27, admitted under Dr. Taylor, and was subsequently under Dr. Pye-Smith and Dr. Hale White, in John, 16, November 5th, 1894.

Yesterday while going upstairs the bannister broke, and he fell backwards and got a bad shaking. To-day at 3 p.m. (he had previously eaten a good dinner), when exerting himself, he suddenly had a most acute pain in upper part of abdomen (right hypochondrium).

Admitted for sudden violent abdominal pain; rigid abdomen. No physical signs in chest. Vomiting and hæmatemesis soon after admission. Perforated gastric ulcer suspected.

Condition on admission.—At 5 p.m. patient was rolling about in agony. Speech almost impossible from shortness of expiration. Abdomen very tender, moving badly, hard and tense in right hypochondrium; thighs flexed. Temp. 103°. *Respiration* very shallow and rapid. *Pulse* fairly good, certainly not wavy or running, but rapid. In ward at 5.30 p.m.—Face drawn with pain, but certainly not "abdominal." He lies on one side —showing no preference for either—with thighs drawn up. Legs and feet seem tender. No jaundice; no rash. No abnormal signs in *heart* or *lungs*. Skin dry, not "pungently" hot. Bowels constipated; abdomen not retracted and not distended; tongue large, flabby, not furred. 8.30 p.m.—A little easier, occasionally dozing. Has vomited two or three times. *Pulse* still rapid (112), full and bounding. *Respiration* 29. Slight occasional cough and rhonchus in front. 11 p.m.—Tubular breathing and bronchophony at right base.

November 6th.—12.30 a.m.—Dr. Taylor found distinct dulness and bronchophony at right base. Patient assisted matters by expectorating a little rusty sputum. 2 a.m.—Patient now comfortably sleeping, lies on left side. Sweats profusely. 11 a.m.—Has slept fairly. Vomited once; more rusty sputum. Much less pain; can talk easily. Abdomen still rigid and tender. Absolute dulness to eighth rib on right. Resonance diminished to spine of scapula. Tubular breathing and bronchophony in eighth and ninth spaces outside angle of scapula. No entry of air below tenth rib. No râles, no rub. Left lung quite clear. *Urine:* No albumen, no sugar, chlorides not diminished. *Sputum:* No pneumococci found.

7th.—Bowels acted with calomel. Tongue furred. Vomiting less. Skin hot and dry. Sputum profuse, blood-stained. Dulness unchanged. Tubular breathing and bronchophony are much more obvious and audible over a larger area, *i.e.* below seventh and eleventh ribs and back to spine. Loud pectoriloquy. A few fine râles audible here after coughing. *Abdomen* still rather rigid, with pain in epigastrium.

8th.—Midnight (12.5 a.m.).—Patient suddenly started up in great pain at upper part of left chest. Sweats profusely at night. Very dyspnœic. Respirations 36. Tongue all over brown sordes, very dry. *Heart:* First sound rough and rumbling. Less absolute dulness at base, where there is now less tubular breathing and bronchophony; no pectoriloquy. Up to fourth rib, however, there is bronchophony. On left, tubular breathing and bronchophony are distinct in axilla, and extend to front and back. Note good.

9th.—Ice poultice has quite relieved pain. Right side note good all over. Small spot of tubular breathing at base. A few râles. Left, marked tubular breathing even to second space in front. Note deficient over small area at base. Moving patient on to right side causes cyanosis and distress. Abundant bloody sputum.

10th.—Signs: *Right lung:* Rub at base; small area of tubular breathing higher up. *Left lung* uniformly dull; tubular breathing and bronchophony up to scapular spine behind and clavicle in front. Patient delirious this evening.

11th.—*Right lung:* Note good; rub gone. No evidence of fluid. *Left*

lung: Loud crackling râles all over. Very little bronchophony and tubular breathing. Patient has been delirious all day, and has been given oxygen. 11 p.m.—Loud rub in left axilla. Dulness slight at both bases, especially left. Heart's impulse in sixth space just external to nipple.

12th.—2 a.m.—Delirium. 10 a.m.—A little better. Skin pungently hot. 8 p.m.—Dr. Pye-Smith heard tubular breathing at angle of left scapula. No rusty sputum for two days; scanty froth only. *Crisis.*—12 p.m.—Patient sweating freely; pain gone; mind perfectly clear; lies on back.

13th.—This morning the temperature is 99·4°; previously it had always been between 102° and 104°. Bowels have acted spontaneously. Urine contains much lithates; no albumen. Both sides in *front* move well, and present no abnormal sounds; *behind* both bases dull. Crackling râles general on left, scanty on right near spine. Tubular breathing near angle of left scapula. Well-marked rub in left axilla. Heart's impulse sixth space in nipple line.

16th.—Temperature up to 100°. Patient very comfortable. Physical signs not materially altered. Right base dull up to seventh rib, left up to ninth. No tubular breathing. Still a rub in lower part of left axilla. Fine crepitations at both bases and in axillæ.

17th.—Since the 13th the temperature has ranged between normal and 101°, being higher in the evening. No rub; less dulness on left side.

18th.—Patch of tubular breathing about eighth rib on right side.

19th.—Left base quite resonant; good entry of air. On right side dull up to eighth rib. Over this area deficient tactile vocal fremitus, air entry and voice sounds; tubular breathing at right base.

20th.—6.30 p.m.—Sudden pain in right side and painful hacking cough. Patient flushed and restless.

21st.—Heart still displaced. Patient better. Appetite good. Signs unchanged.

23rd.—Some tenderness over right base. *Signs:* Dulness at right base not so marked. No entry below ninth rib. Distant tubular breathing and bronchophony over seventh, eighth, and ninth ribs near spines.

28th.—Patient seems much worse. Drowsy; voice weak; much cough; frothy thin sputum. Pneumococci found in it. *Signs:* Dulness extending.

29th.—Exploring needle in ninth space drew off pus containing pneumococci. Aspiration decided on, as no staphylococci or streptococci are to be seen.

30th.—The temperature has been constantly raised, usually about 100° in the morning and 101° in the evening. Aspiration ʒix; much glairy lymph since.

December 1st.—Temperature normal. Right base more resonant; entry bad.

6th.—Temperature rising again. More resonance; entry still bad.

7th.—Patient uncomfortable and restless; cough bad.

8th.—Œdema at site of puncture.

11th.—Drainage from ninth space. Half a pint of pus escaped. Cavity apparently small. Floor felt to be thickened lung. From this time he did well. He left the hospital early in January.

Summary of Pulse, Respiration, and Temperature.

November 5th—12th.—Pulse 110—148, resp. (5th—8th) 28—52; (9th—12th) 40—64, temperature usually between 102° and 103°.

13th.—Crisis. Temperature fell from 103·4° to 99·4°, pulse to 92, resp. to 32.

15th—30th.—Evening rises of temperature began on the 15th, and they gradually increased day by day till 30th (day of aspiration). The evening temperature was often over 102°; morning temperature a degree or so lower. Pulse 80—104, resp. 24—40.

December 1st and 2nd.—Temperature under 99°, pulse 72—88, resp. 20—32.

3rd—10th.—Evening rises and morning falls of temperature began again. Morning temperature about 99°, evening temperature 100°—103°; pulse 76—100, resp. 22—32.

11th (operation)—18th.—Temperature about normal, pulse 72—100, resp. 18—28.

Case 3 (1893).—Eliza P—, æt. 18, admitted into Miriam 30th December, 1893, under Dr. Pye-Smith, subsequently under Dr. Hale White. Death on January 10th. She came in for pain in the chest and difficulty of breathing. At 9 p.m. on December 29th she had rigors.

On admission.—Much dyspnœa. Temp. 103·4°; pulse 140; resp. 50. Face flushed. Short dry cough; much rusty sputum. Dulness at left base up to the level of the ninth rib. Over dull area no vocal fremitus, no breath-sounds, no tactile fremitus. Œgophony and bronchial breathing at angle of scapula, and tubular breathing same spot. Right side and front of left chest healthy. Except for hæmic murmur rest of body healthy.

December 31st.—Left chest aspirated, but only ʒiij of purulent fluid were withdrawn; physical signs unchanged.

January 2nd.—The physical signs are unchanged, except that distinct tubular breathing can be heard over the left base; it is, however, faint. Urine contains a trace of albumen.

3rd.—As the dyspnœa was great, a needle was passed into the left chest, but the fluid drawn off was only turbid; there were not any signs of increase of fluid.

4th.—Physical signs over the left lung the same, but to-day there is distinct tubular breathing over the right at the spine of the scapula, and here there are some consonating râles.

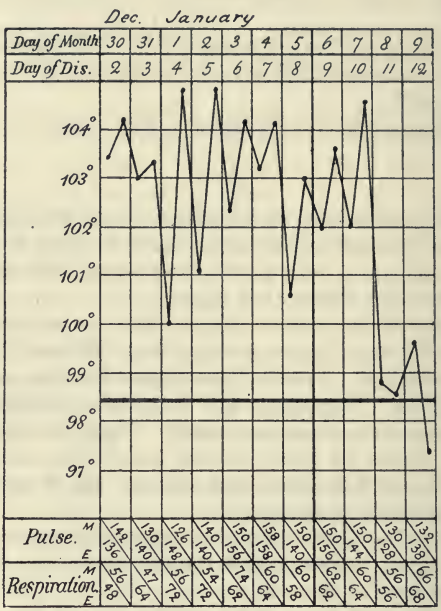
6th.—There is dulness on the right below the spine of the scapula, and over the dull area there are bronchial breathing, bronchophony, and consonating râles. The physical signs on the left are almost unchanged, but on the whole the bronchial breathing is more distant and the dulness is more absolute.

9th.—The chest is, on the left, dull to the spine of the scapula, with distant tubular breathing and distant crackling râles. Right side dull up to the right side of scapula, with bronchophony, bronchial breathing, and consonat-

ing râles. Temperature this morning subnormal, and the patient seems better in every way.

10th, 4 a.m.—Patient died quite suddenly. The treatment consisted of brandy; Injectio Morphinæ Hypodermica gr. ¼ to give sleep; Mist. Ammoniæ ʒj t. d. s.; sponging whenever the temperature was over 104°.

Autopsy.—Recent right pleurisy. Much thick curdy lymph over the left pleura, and its cavity contained 20 oz. of thin pus. *Right lung:* The whole of the lower lobe was solid from lobar pneumonia, but it was tougher than usual. The left lung was compressed, and therefore it was difficult to recognise in it the signs of pneumonia.



Case 4 (1893).—John R—, æt. 8, admitted into John Ward February 4th, 1893, under Dr. Hale White, and subsequently under Dr. Goodhart; clinical clerk Mr. F. P. S. Cresswell. Died March 3rd.

History.—Always subject to coughs.

January 30th.—Came home from school with shivering and vomiting (he had got wet through four days previously).

February 2nd.—Another attack of shivering, and took to bed with pain in right chest and back, also hacking cough.

3rd.—This and last night he slept little; sweating profusely; cheeks flushed. Sputum pinkish to-day.

Condition on admission (February 4th).—Half-starved; pale; cheeks flushed; much pain. *Lungs:* Both sides move equally. Right, behind,

dull up to eighth rib, tubular breathing, bronchophony. Left, in axilla, dull up to fourth rib, tubular breathing; behind, dull at base, tubular breathing, bronchophony. *Circulation:* Pulse 140, small. *Urine:* Febrile; no albumen. *Tongue:* White fur. *Bowels* constipated.

Treatment.—Ice-poultice. Mist. Sp. Vini Gallici ʒss 3tis horis; milk; chloralamide gr. x h. n.

5th.—Faint rub in left axilla, traceable round to base. Patient lies on left side to minimise the pain. He has slept a good deal to-day.

6th.—*Signs:* Rub well marked; loud to-and-fro pericardial rub. Patient much worse to-night; very restless and delirious. Pulse very feeble, small, irregular, 100.

Treatment.—Ice poultice omitted; linseed and mustard substituted. Calomel gr. iii having failed, Enema Saponis given; Mist. Sp. Vini Gallici ʒj, 3tis horis; Pulv. Opii gr. $\frac{1}{6}$ 6tis horis.

7th, 2 a.m.—Temp. 97·8°; pulse 116, very feeble, irregular and running; resp. rapid (60); no lividity; no evidence of right-sided dilatation. Much sweating; fair amount of sleep. Noon.—Pulse stronger; child better, still sleeping a great deal. *Signs:* Pericardial rub hardly audible. Right lung, in front, few râles below nipple. Left lung, in axilla, loud râles, smaller ones at base. Pleuritic rub still audible in front. Tubular breathing only in sixth space in axillary line. Back not examined.

Treatment.—Tinct. Digit., Tinct. Nucis Vom., āā ♏v 4tis horis.

8th.—Bowels opened twice this morning; loose. *Signs:* Complete dulness over left base and left axilla, running into heart dulness, which reaches a little above third rib in front. Impulse in fifth space one inch outside nipple line. Pericardial rub only audible at apex, not at base.

9th.—Patient was fairly quiet all night. Digitalis omitted, pulse having become irregular and intermittent; brandy substituted. *Signs* as yesterday. Colour of face keeps good, and pulse has improved since digitalis was omitted. Resp. 40.

10th.—Poultice and opium omitted, and patient slept well. Patient can lie on either side now, though respirations are still rapid. *Signs:* Rubs remain as before.

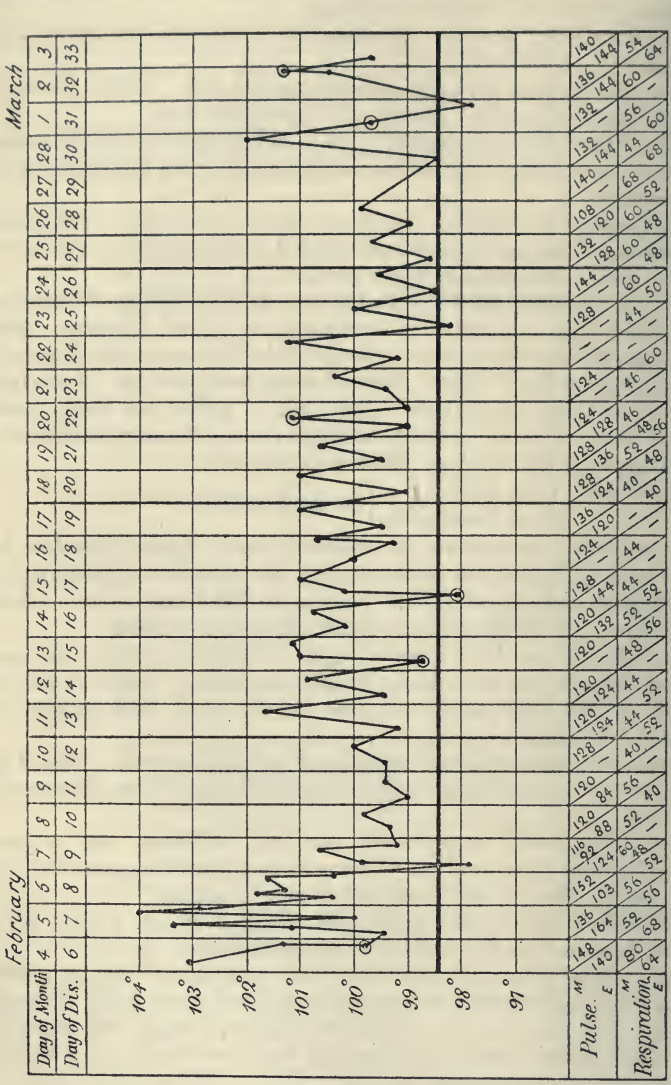
11th.—Slept well; complains of pain and tenderness over stomach. Temp. 100° last night, resp. 52. *Signs:* Rubs still audible, pericardial much diminished; râles all over bases; still tubular breathing.

13th.—Pain much better; patient feels hungry. *Signs:* Dulness behind at both bases and tubular breathing; no pericardial rub audible.

15th.—Pulse more rapid and dicrotic, 144; temperature up again to 101°. *Signs:* Dulness as before; no diminution of breath-sounds either side; râles all over both bases and some tubular breathing; pleuritic rub fainter on left, still marked on right. Patient is a little more restless, and does not sleep so well; is still sweating freely.

20th.—Patient better and more comfortable. *Signs:* Loud tubular breathing at left apex in front; breath-sounds do not seem so much diminished in axillary line as in second and third spaces nearer the sternum. Otherwise signs remain as before.

23rd.—Condition stationary. *Signs:* Little tubular breathing at apex; diminution in breath-sounds less marked; impulse diffuse in fifth and sixth spaces, 1½ inches outside nipple line; loud rub at left apex in front, also



over front of right chest; behind, the rub is still present at left base, with loud tubular breathing and crepitant râles at right base; there is only a little tubular breathing in sixth space.

25th.—*Signs* as before; temperature gradually going down; patient sleeping and eating well and improving.

28th.—*Signs:* Pleuritic rub absent both sides; behind, dulness continues up to seventh rib, on both sides crackling râles and tubular breathing as before; in front, at left apex, breath sounds are much louder than previously, there are râles all over; respirations average 54 to-day.

March 2nd.—Patient not so well, perspires a great deal. *Signs* as before; pulse dicrotic, 144; impulse diffuse; resp. 50.

3rd.—Last night patient had a little sickness; looked much worse; restless in night, and dyspnœa increased. He suddenly got worse, and died at 8 this morning.

Autopsy.—Twelve ounces of pus in right chest. Both lungs much compressed, especially left, which was pushed right up against spine. Right upper lobe and part of left upper lobe were alone crepitant. No evidence of pneumonia except that upper half of left upper lobe was solid and red, and adherent to chest-wall in front. Right pleura covered with thick lymph; recent pleurisy all over left side. *Pericardium* much thickened, contained twenty-three ounces of pus, it occupied most of left chest, greatly compressing lung. *Heart:* covered with thick yellow lymph; muscle bad; apex of left ventricle adherent to pericardium by strong fibrous band half an inch long, explaining forcible impulse before death in spite of effusion.

On posterior flap of mitral was a small vegetation, blood-stained and pretty firmly adherent.

Case 5 (1893).—Charles F—, æt. 22, a carman, admitted into John Ward under Dr. Hale White, January 17th, 1893, died January 20th. Clinical clerk Mr. W. J. Harris. Not habitually a hard drinker, but got drunk on Christmas day when he was married.

January 14th.—Going home from work had cold shivers and felt very poorly. He retched during the night.

15th.—Bowels opened three times after taking a pill.

17th.—He has got worse ever since.

Condition on admission.—Face flushed and cyanosed; lips purple; eyes sunken. Great dyspnœa. Respirations 52, short and shallow. Cough frequent, loose. Sputum bright red, mucoid. *Signs:* In front, right side moves badly, tubular breathing at apex; behind, note deficient at both apices. Right lung resonant below sixth rib, tubular breathing at apex. Left lung dull the whole way down. Loud tubular breathing at base, becoming feeble at apex. Bronchophony. *Circulation:* pulse 152, full and bounding. Heart-sounds normal. *Urine:* 1024, acid, dark, albumen present, no blood. Pulse 132; resp. 52; temp. 103·2°.

Treatment.—Ice poultice to left base. Brandy ʒiv, increased to ʒvj; Pulv. Opii gr. j t. d. s.; Inject. Morph. Hypoderm. gr. $\frac{1}{6}$, 2 a.m.; milk; yolk of two eggs.

Progress.—January 18th.—Feels slightly better. Temp. 100°. This was thought to represent the crisis. *Signs:* Right apex in front, no tubular breathing to be heard. In the afternoon pulse 116, resp. 56, temp. 99·8°. In the evening pulse 126, resp. 58, temp. 102·8°.

19th.—Ice poultice omitted. To-day respiration is shallower, and pulse smaller. He has several injections of morphia to procure sleep. *Signs:* Both bases dull, with tubular breathing. Dulness extends on left up to spine of scapula, on right not so high. Morning, pulse 130, resp. 62, temp. 100°. Evening, pulse 148, resp. 69, temp. 103·2°.

20th.—Pulse and respiration failing since midnight. Hot coffee ʒvj *per rectum* appeared to effect some improvement. 5 a.m., died suddenly.

Autopsy.—Both lungs solid (red hepatisation) except anterior portions of both upper lobes. Recent pleurisy on both sides, ʒxvj of turbid semi-purulent fluid in the right chest, which, however, did not appear to have compressed the lung. In left chest at back of lower lobe was a thick mass of lymph and pus, which indented the lung to the depth of half an inch, forming a small localised empyema. No sign of pericarditis. Kidneys congested, 11 oz.

Case 6 (1893).—Lewis H—, æt. 46, admitted under Dr. Hale White, October 20th, 1893. Clinical clerk, Mr. H. A. Moffat. He came in for pain in the chest and general illness. The only thing of importance in the previous history is that he is a reformed drunkard.

Present illness.—On October 16th he suddenly felt ill with a severe pain in the right side, worse when he drew a breath. He went home and took to bed, where he has remained till admission. He shivered on October 18th, then the cough began and the sputum became rusty.

On admission.—Temp. 101°, pulse 108, resp. 50. There is a very loud rub over the lower part of the right chest. It can be easily felt with the hand. This side is dull and tender below sixth rib. Not much air enters, but then the movement is very slight, There is bronchophony. Left side normal. Sputum rusty. Herpes on lips. The cardiac apex is in the normal position, and no murmurs can be heard. No albumen. The diagnosis was pneumonia with very extensive pleurisy.

October 23rd.—The evidence of pneumonia has remained as before; the rub is still very loud. Bronchial breathing and fine crepitations can now be heard on the right side, behind. To-day he is better as the temperature has fallen, but he is clearly a very severe case.

24th.—Is much better. There are now loud moist râles over both chests.

25th.—He is not quite so well. There are still loud râles in both lungs. The pleuritic rub can still be heard on the right, where also bronchial breathing is audible from below the fifth rib to the base, and the air enters here fairly well. This improvement in the entry of air and the presence of the râles made us think there was not much fluid. No cardiac murmurs were heard.

28th.—The patient has distinctly improved the last few days. The right lung is still dull as high as the spine of the scapula. The air enters well on both sides. Rhonchi and râles are heard over both sides, especially on the right and over the dull area. Sputum still slightly rusty. He is able to lie down and to breathe comfortably. Heart-sounds normal.

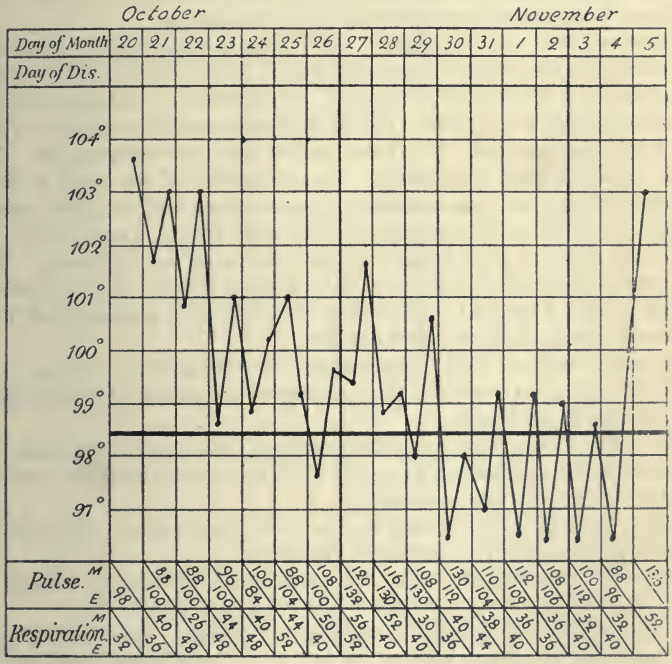
30th.—Is much better. Physical signs as before. Temperature normal or subnormal all day.

November 2nd.—Temperature yesterday was once 99·6°; to-day its

highest point was 99·2°. The physical signs are the same, except that the râles are not quite so distinct.

3rd.—The highest temperature was 99°. There is a loud rub in the right axilla, otherwise the physical signs are the same. For the last three days the patient has had severe diarrhœa.

5th.—At 2 a.m. this morning the patient had a severe attack of dyspnœa. The pulse was slapping, but no cardiac murmurs could be heard; the second sound was absent. An exploring needle was put in the eighth space on the right side in the post-axillary line, but no fluid was found. The patient gradually got worse and worse, and in spite of all efforts to revive him died at 6 a.m.



Post-mortem examination.—An empyema was found on the right side, bounded in front by the right nipple line, behind by the right mid-axillary and corresponding to the fourth, fifth, and sixth ribs. It contained five or six ounces of thick pus. The exploring needle had passed behind it. Lymph over nearly all the right lung. Left pleura healthy. The upper right lobe was œdematous; the middle lobe was compressed by the empyema; the lower was solid, and on section showed several abscess cavities containing foul greyish-yellow pus; the smallest was the size of a pea, the largest eight times as large. They were close to the empyema, and had irregular shaggy walls, with broken-down lung tissue around them. Left lung

emphysematous, otherwise healthy. *Heart:* On the left anterior cusp of the pulmonary valve was a warty mass about the size of a pea, which broke down on being touched, and turned out to be an abscess. The tricuspid was healthy. The mitral valve had a few fine vegetations on it. There was a large, soft, blood-stained vegetation on the ventricular surface of the anterior semilunar valve of the aortic orifice, easily breaking down; it was the size of a large horse-bean. In the middle of it was a perforation through the valve.

Case 7 (1893).—L. E. C—, æt. 23, admitted into Guy's Hospital January 8th, 1893, under the care of Dr. Washbourn, and subsequently under Dr. Hale White. On January 6th he was suddenly seized with a rigor. Next day he suffered from pain in the right side, attended with fever. He was seen by a medical man, who said he had pneumonia.

On admission.—He is obviously very ill, and in much pain. The pain is increased by a troublesome cough and by any effort to move. The respirations are rapid, shallow, 50 per minute. The sputum is scanty, and consists of mucus, which is not rusty. The tongue is covered with white fur, and the bowels are confined. The pulse is 133, weak and compressible. The urine contains a trace of albumen. An examination of the chest revealed dulness at the right base behind with crepitations and deficient breath-sounds. A pleuritic rub was audible in the right axilla. Temp. 102·6°.

For the next four days the general condition remained unchanged. The temperature was high, and on one occasion reached 103·4°. The respirations varied between 40 and 60. The base of the right lung remained dull, loud râles and occasional rhonchi were audible.

January 13th.—A slight improvement in the general condition was observed. The pain was less, the temperature lower, and the respirations had sunk to 30 per minute.

The improvement continued, and on January 16th the pain and cough had disappeared, the respirations were only 22 to the minute, the pulse was 80, and the temperature nearly normal.

17th.—Resp. 20. The patient was considered convalescent. The physical signs in the lung, however, remained unchanged.

18th.—The temperature, which had never been quite normal, went up to 101°.

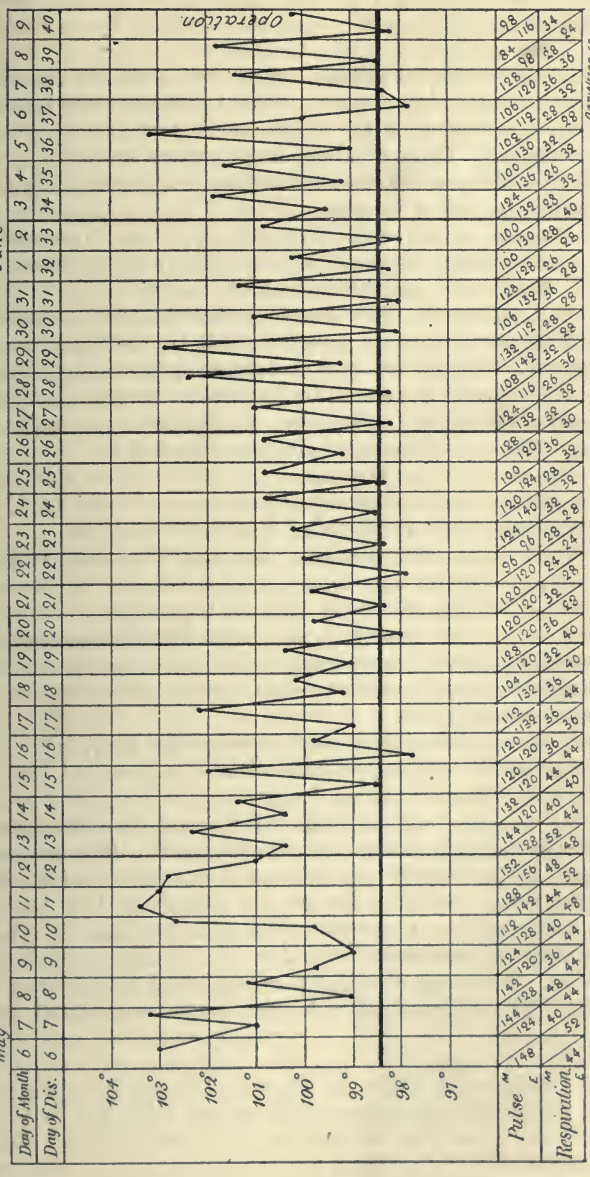
During the next five days the temperature oscillated between 101° and 102°, and the respirations between 22 and 28. The physical signs, which had never cleared up, gradually pointed definitely to the presence of fluid in the chest.

24th.—As an exploring needle revealed pus an anæsthetic was given, and a pint of pus evacuated.

From this time the temperature began to fall; the patient made a rapid recovery, and when last heard of was in excellent health.

The pus was examined microscopically, by cultivations and by inoculation experiments, and was found to contain the pneumococcus of Fraenkel.

Case 8 (1893). *Double pneumonia; empyema.*—Walter R—, æt. 6, admitted into Stephen Ward under Dr. Hale White, May 6th, 1893; clinical


May
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clerk Mr. M. Parry Jones. Patient came in for fever, and pain in the right side. He has always been strong and healthy.

Present illness.—On May 1st, in the evening, he felt ill, vomited, and complained of pain in the side. As he got worse and had great difficulty of breathing he was brought to the Hospital.

On admission.—Skin hot and dry. Temp. 103°, pulse 148, resp. 44. Left chest does not move so well as right. Marked dulness at both bases behind, with tubular breathing from the eighth to the tenth rib on both sides. Râles at both sides. Urinary and circulatory systems normal.

May 10th.—Well-marked tubular breathing and bronchophony over the greater part of the back of the chest.

13th.—There is now a systolic bruit at the junction of the seventh rib with its costal cartilage, and there are râles in the front and lower part of the chest. The physical signs at the back are unaltered.

16th.—Temperature was below normal for the first time since admission. The heart's impulse is displaced inwards towards the sternum; the systolic bruit is fainter. There is well-marked dulness behind from the angles of the scapulæ downwards, and also in the left axilla. Tubular breathing left side at level of seventh and eighth ribs. Diminished tactile vocal fremitus both bases. Urine, no albumen. Left disc a little blurred.

23rd.—The physical signs have altered very little. There is still dulness, tubular breathing, and bronchophony at both bases, and the faint systolic murmur is audible. But he complains of pain in the left side.

31st.—There is very little change in the patient's condition, except that, on the whole, the physical signs are less marked, although the temperature had for the last few days been rising higher in the evening.

June 3rd.—As the temperature is still raised each day, pus was thought to be present, and as the dulness was more marked and the entry of air deficient in the left axilla, a needle was inserted in the seventh left intercostal space, but no pus was found.

6th.—As from the temperature it still seemed certain that pus was present, a needle was inserted into the left axilla after chloroform had been given, and in the fourth insertion pus was found.

9th.—The empyema was laid open, but it was a remarkably small and localised collection of pus, for only ʒss was discovered. After the operation the patient's health quickly improved, but it was four weeks before the temperature became steady. He left the hospital on July 15th, 1893. For the first week the temperature was much the same as before the operation, then for three weeks it was about 100° in the evening and 98° in the morning. During this time the pulse was rapid in proportion to the temperature; the rapidity of respiration was, on the whole, slightly in excess of that of the pulse.

Case 9 (1893).—Lizzie H—, æt. 30, admitted into Mary Ward, under Dr. Hale White, 29th September, 1893; clinical clerk, Mr. R. O. Moon. She came in for pains in the left shoulder. Her mother and her sister have died of phthisis.

Previous history.—A year ago she had rheumatic fever very badly.

Since then she has at times had great difficulty in getting through her work as a domestic servant.

Present illness.—On September 23rd patient was taken ill, having rigors and a bad headache. On 26th she had to remain in bed in a drowsy condition. On 27th she again had rigors.

On admission.—She lay on the left side, and had all the signs of left basal pneumonia with a moderate amount of effusion; the sputum was rusty. There were various pains about the body, especially in the joints, but since her rheumatic fever she had been liable to these. The cardiac apex is in the normal position, but a well-marked systolic murmur, apparently due to mitral regurgitation, is audible there. The cardiac dulness is not increased, and at the base a distinct pericardial rub may be heard. There is a trace of albumen in the urine. During the first fortnight or three weeks of her stay in the hospital she suffered much from diarrhœa, but this was ultimately controlled with compound catechu powder.

October 7th.—The sputum is still rusty. The signs in the left chest are chiefly those of a moderate amount of effusion. The pericardial rub is still audible, but the area of dulness is slightly in excess of normal.

11th.—The pericardial rub has disappeared. The signs indicate that the effusion in the left chest is slowly decreasing, and the sputum is only very slightly rusty.

18th.—The pericardial rub has from time to time been audible during the past week, but the fluid in the left chest is apparently being slowly absorbed. The sputum is not rusty, and the patient has improved much in health.

November 1st.—The patient, who has been slowly improving in all respects, now gets up after dinner.

6th.—The patient now complains of pain at a spot about three inches below the angle of the left scapula, and as the temperature has gone up again she was put to bed. There is no alteration in the physical signs in the left chest, which are those of thickened pleura. The pericardial rub cannot be heard.

8th.—The temperature has continued up since the last note. The diagnoses of empyema and malignant endocarditis were discussed. A needle inserted into the left chest failed to withdraw pus.

13th.—The temperature still continues raised, and a needle inserted a little below the last point withdrew pus. In the evening this empyema was laid open, and it was found to contain only ʒiv of pus. From this time the patient slowly improved without a bad symptom.

Temperature.—The chart is too long to print, and can be summarised.

On admission, temp. 103°, pulse 120, resp. 28.

September 30th.—Morning temp. 101·6°, pulse 100, resp. 24. Evening temp. 102·4°, pulse 100, resp. 28.

October 1st—6th.—Morning temp. 100°—101°, pulse 90—108, resp. 24—28. Evening temp. 102°—103°, pulse 100—120, resp. 24—36.

7th—11th.—Morning and evening temperatures about 100°, pulse 84—100, resp. 24.

12th—15th.—Morning temperature about 100°, pulse 99—100, resp. 24. Evening temp. 100·2°—101·8°, pulse 92—104, resp. 20—24.

15th—November 6th.—Morning temp. 98°—99·4°. Evening temp. 99°—100·4°.

6th—12th.—Temperature gradually mounted, till on November 12th morning temp. 101·2°, evening temp. 103·8°.

After the operation, on November 13th, temperature fell to normal, and remained about normal till discharge.

Case 10 (1893).—Bertram L—, æt. 20, admitted April 26th, 1893, under Dr. Goodhart. Clinical clerk, Mr. A. Salter. Discharged July 5th, 1893.

History.—Has had bronchitis every winter since influenza four years ago.

April 24th.—This morning while at work patient was suddenly taken with violent shivering, faintness, shortness of breath, sensation of intense heat. Dyspnœa growing worse he went to bed, and there had another rigor.

Condition on admission (April 26th).—Strong muscular man. Face drawn and anxious; pale with exception of cheeks, which are intensely flushed; eyes strikingly bright; skin dry and very hot. Urgent dyspnœa, compelling him to sit up in bed. Intense pain in left chest, and occipital headache. Sputum small in amount, thick, rusty. *Lungs.*—Chest well formed, sides move equally; respirations very shallow, without bulging or retraction of intercostal spaces. Left chest, dulness at left base up to eighth rib; breath sounds diminished; distant tubular breathing (but loud at upper limit of dulness). Tactile vocal fremitus, and voice-sounds much increased. Râles all over left chest except apex in front. Dulness at right base below tenth rib behind. *Circulation:* Heart-sounds appear distant; best heard to right of sternum. Pulse very rapid, small and feeble. *Alimentary system:* tongue thickly coated, except at sides and tip. *Urine:* 1038, very dark, strongly acid, deposit of lithates, total absence of chlorides, trace of albumen.

27th.—Still much distress. *Signs:* Dulness at right base, now up to eighth rib; dulness at left base, now up to sixth rib. Breath-sounds absent below eighth rib on right, and below seventh rib on left; diminished above this. Otherwise same.

28th.—Four leeches applied over seat of pain in left axilla, ice-bag later, giving much relief. Pil. Doveri. *Signs:* Still loud bronchophony at bases.

29th.—Crisis this morning. Temperature fell to 100·2°, with much diarrhœa and sweating. Resp. 40, pulse 116. Patient has lost all pain, and breathing is much easier.

30th.—Diarrhœa all day, temp. 102° in evening. *Signs:* Dulness now up to fifth rib on left.

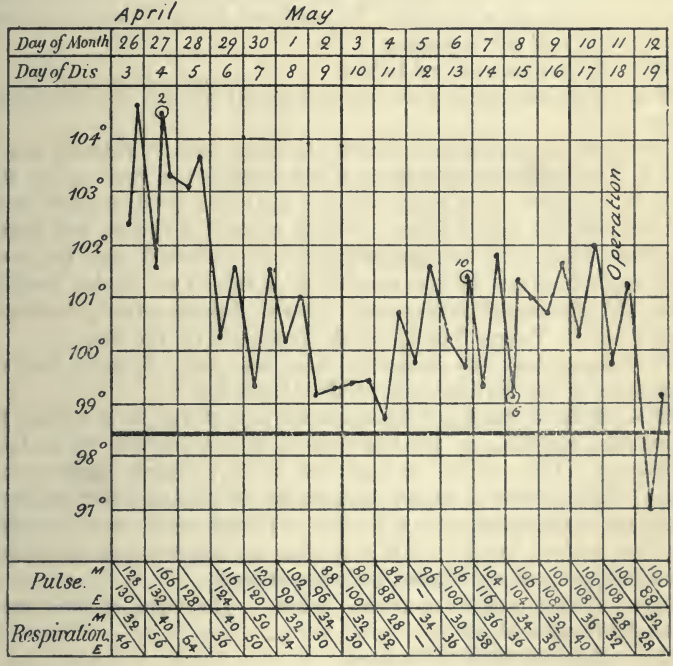
May 1st.—*Signs:* Heart-sounds now *only* heard on right of sternum. Heart impulse lost to sight and touch. Absolute dulness up to third rib behind on left; partial dulness for two inches higher. Absolute dulness up to fifth rib behind on right; partial dulness for one and a half inches higher. In front dulness below third rib on right; dulness below second rib on left. Total absence of breath-sounds on both sides up to sixth rib; tubular breathing above, though not at apices; loud bronchophony accompanies the tubular breathing, and there is some ægophony at scapular angles. Tactile vocal fremitus absent at bases, diminished above.

3rd.—*Signs:* Left, behind, absolute dulness only up to seventh rib; in

front, absolute dulness only up to third rib: right, behind, absolute dulness only up to ninth rib; in front, absolute dulness only up to seventh rib. Breath-sounds have correspondingly gained, and can be heard everywhere, except at extreme bases. Distant tubular breathing, ægophony only at angle of left scapula. Bronchophony only over upper half of left chest behind. Tactile vocal fremitus absent over lower half of left chest behind; increased over lower half of right chest behind.

4th.—No tubular breathing, but well-marked ægophony as before. Loud râles over both chests. Heart-sounds now heard to left of sternum. Diarrhœa has ceased. Temp. 98·8°, pulse 92, resp. 38.

7th.—*Signs:* Rub and sharp pain under left nipple.



8th.—*Signs:* Resonance normal on right; dulness as before on left, breath-sounds here much diminished, absent below; pus obtained from just below angle of scapula.

11th.—*Operation.*—Eighth rib resected, nearly four pints of thick pus escaping.

15th.—Discharge sour and profuse; some diarrhœa yesterday, easily controlled.

28th.—Discharge much less; temperature normal.

June 4th.—Patient gets up.

9th.—Discharge still profuse.

19th.—Discharge slight, still sourish; tube removed thirty-nine days after operation.

25th.—Temperature suddenly gone up to 101·6°; slight rigor, headache and malaise; cavity syringed and considerable amount of pus evacuated.

27th.—Probe passes in two or three inches; tube reinserted.

July 2nd.—Scanty discharge; tube removed again.

5th.—Patient is gaining flesh and seems very well. *Signs:* Still a small area of impaired resonance in region of wound. He goes out to-day.

Case 11 (1893).—Thomas S—, æt. 42, admitted into Philip, under Dr. Pye-Smith, September 15th, 1893.

September 10th.—Was walking down to hop-picking this evening when a sharp pain came on in left side. He continued to walk, however, through the night, though pain increased.

11th.—Went home, but not to bed.

15th.—Pain has steadily got worse, and patient felt very ill; he has had no rigors.

Condition on admission.—Patient has severe pain in abdomen and left side of chest; urine contains albumen but no deposit or casts; sp. gr. 1020. *Lungs:* left chest moves badly. Tactile vocal fremitus diminished at base and in axilla, this area is tender; dull up to angle of scapula and sixth rib in axilla; voice- and breath-sounds diminished in this dull area; few râles in axilla and a pleuritic rub over seventh rib in axilla; no tubular breathing. *Pulse* 124, regular, soft, rather small. *Heart:* dulness normal; faint apical systolic bruit. Tongue furred; bowels constipated for two days.

Treatment.—Left side strapped. Mist. Am. Acet. Injectio Morphinæ Hypodermica $\frac{1}{6}$ gr. on admission; Pulv. Doveri h.n.

16th.—Pulse continuing rapid and small, patient was put on brandy ʒiij; strychnine, digitalis, and Pil. Col. c̄ Cal. h. n. and Haustus Sennæ Co. m.

Progress.—18th.—Pulse stronger and slower; sputum slightly rusty. *Signs:* Faint tubular breathing at upper part of left axilla and consonating râles and bronchophony, below this voice- and breath-sounds much diminished and râles are few; behind at left base, voice- and breath-sounds much diminished at upper part of dull area, quite lost at base.

20th.—Patient altogether much better. The signs of fluid extend up to eighth rib behind.

21st.—Rub over third rib in front.

23rd.—Breath- and voice-sounds and tactile vocal fremitus slightly improved.

26th.—Chest very dull, breath-sounds very faint; needle put in to-day drew off pus.

28th.—*Signs:* Dulness in whole of axilla now and up to sixth rib behind.

29th.—*Operation.*—ʒvj to ʒvij evacuated through third space behind; tube put in.

October 3rd.—Discharge now slight.

5th.—Discharge equals about ʒij.

9th.—Dressing still saturated with pus.

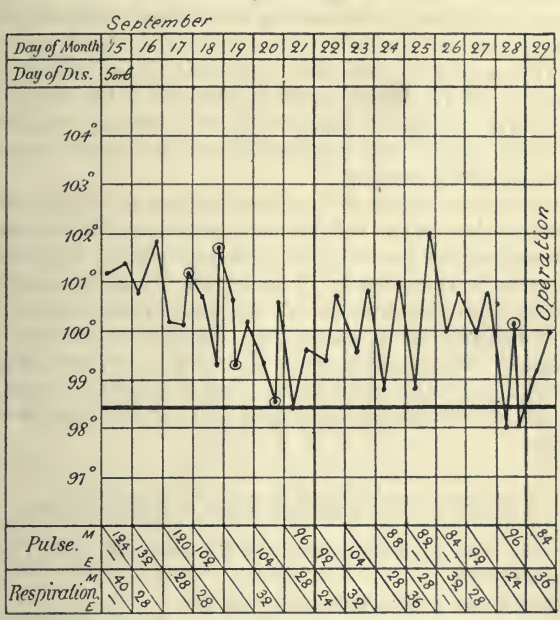
14th.—Dressing still saturated with pus; temperature normal.

18th.—Tube removed.

21st.—*Signs:* Area round wound dull; breath-sounds diminished; some râles; friction sound two inches from wound.

27th.—Area round wound clearing up; moist sounds heard on auscultation.

November 1st.—Patient discharged cured.



Case 12 (1892).—Charles R—, æt. 23, worker in hides, admitted into John Ward March 31st, 1892, first under Dr. Hale White, and subsequently under Dr. Newton Pitt.

History.—None of tubercle in family. Patient always temperate; no previous serious illness.

March 27th.—Sudden pain in right side, shooting to left shoulder; feverishness; no rigor. Stayed in bed till he came to the Hospital.

Condition on admission (31st).—Marked dyspnœa; resp. 64; pulse 132, failing; no great cyanosis. Sputum rusty. Dulness over both bases. Loud tubular breathing. Inspiratory and expiratory metallic râles.

Progress.—31st.—Patient was delirious this night, and succeeded in escaping from the ward into the colonnade.

April 1st.—Sputum muco-purulent and viscid. Right chest had a doubtful rub behind; dulness up to spine of scapula; some bronchial breathing.

2nd.—Patient better. While yawning this morning he dislocated his jaw. It was easily replaced.

3rd.—Better still. Expectoration now copious and frothy; no longer rusty. Rapidity of respiration varies greatly.

6th.—Temperature normal; sweating still profuse.

8th.—Needle put in at eighth space on right; turbid serum drawn off. The right side is still very dull, but bronchophony and bronchial breathing are present over it.

16th.—Expectoration still profuse; purulent. *Signs:* In right posterior axillary line, up to angle of scapula, dull on light percussion, tympanitic with strong percussion; breath-sounds here almost amphoric. Diarrhœa continues.

21st.—Voice-sounds increased above right fifth rib in front. Dulness here extends up to fourth rib, having moved up one space in two days.

27th.—Pain just outside right nipple. No rub; breath-sounds diminished.

29th.—Needle put in ninth space withdrew thick creamy brownish pus. Expectoration entirely purulent.

May 2nd.—Patient advised to have operation, but refused and went home. *Signs:* Same marked dulness and percussion-resonance with complete absence of tactile vocal fremitus over whole of right lung behind. No breath-sounds whatever except at root of lung. Voice-sounds diminished except at root. Œgophony about ninth rib in axilla. *In front* dulness absolute in fourth space; note impaired for an inch above. Marked bronchophony in fourth and fifth spaces. Breath-sounds harsh all over front. There are signs of a local pneumothorax in the right axilla. *Left lung* healthy except fine râles at base. *Heart's impulse* in sixth space half an inch external to nipple; sounds faint.

Summary of Temperature, Pulse, and Respiration.

March 31st.—On admission temp. 101·6°, pulse 132, resp. 70; evening temperature 103·6°.

April 1st.—Temp. 100·2°, pulse 128, resp. 68.

2nd.—Morning temp. 102·2°, pulse 128, resp. 26; evening temp. 98·8°, pulse 120, resp. 40.

3rd.—Morning temp. 102·2°, pulse 108, resp. 36.

4th.—Morning temp. 98·2°, pulse 88, resp. 36.

4th—10th.—Temperature irregular, ranging between normal and 100°; pulse about 100; respirations varying between 36 and 56.

From April 10th the morning temperature was about 99°, the evening temperature about 100·6°; pulse between 80 and 100; but the respirations were nearly always over 30, and often as high as 48.

Case 13 (1892).—Herbert W—, æt. 25, admitted on March 1st, 1892, under Dr. Hale White in John 6. Clinical clerk, Mr. H. E. Durham.

History.—March 1st.—Patient is said (he is deaf and dumb) to have been seized with a rigor this morning. He had another in the surgery.

Condition on admission.—Signs: Pleuritic rub left side of chest in axilla with a patch of dulness the size of the palm of the hand, and bronchial breathing and bronchophony.

Treatment.—Ice poultice.

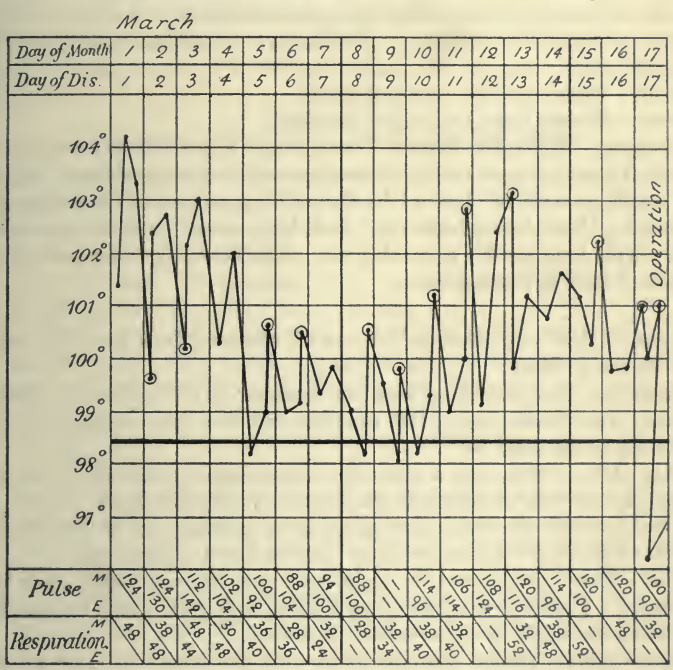
7th.—Patient wants to get up. The lower part of the left chest is dull.

14th.—*Signs:* Great tenderness in left axilla. Total dulness and sense of resistance below halfway between spine and angle of scapula, slight rather tubular breathing sounds above dull area. A little entry of air in left lung in front at left first space, none below. Temperature been rising high the last three nights.

15th.—Needle drew off pus from left axilla.

17th.—Three pints twelve ounces of pus let out, and rib resected.

April 11th.—Patient went home. Wound open, discharge slight.



Case 14 (1891).—Thomas H—, æt. 50, woodheaver, admitted under Dr. Perry, in John 11, May 4th, 1891; death May 24th. Clinical clerk Mr. Donelly.

History.—Has been a very strong man. Influenza last year, not severely. Three weeks ago came into Samaritan Ward with a crushed hand. Last Monday went out not feeling very well. Last Thursday, April 30th, rigors came on, and lasted off and on all day. Friday pain in side, and he came in on May 4th.

Condition on admission.—Sputum copious, frothy, slightly rusty. Complete dulness from angle of scapula downwards on right side with loss of breath and voice sounds. Tubular breathing above dulness, and in axilla. Left lung a few râles and rhonchi. No rub to be heard. Resp. 40, pulse 110. *Heart* normal. *Urine* normal.

May 5th.—Much pain, two leeches applied.

8th.—Needle put in fifth space on right side, no fluid found.

10th.—Seems much better; a little diarrhœa. Clear fluid found to-day.

12th.—Physical signs as before. Patient drowsy, not so well.

15th.—Patient very drowsy, sallow complexion. Entry of air more deficient.

17th.—Patient has a difficulty in swallowing, the act bringing on cough. There was some pain over fourth right costal cartilage.

20th.—Cough still very bad. Dulness up to third space on right; left normal.

23rd.—Pulse very bad. Patient dying.

24th.—Patient died at midnight last night.

Autopsy.—Right chest contained seven ounces of pus between base of right lung and diaphragm, extending "towards anterior and outer sides of lung, but not coming to surface." Lower lobe of right lung compressed and airless. Pus in tubes. Upper lobe œdematous. Left lung normal, but recent adhesions between the lung and the pericardial sac. *Heart* covered with lymph; eight ounces of fluid in pericardium.

Case 15 (1891).—Elizabeth M—, æt. 28, admitted June 2nd, 1891, under Dr. Taylor, in Mary.

History.—Was recovering from an operation (by Mr. Howse in Charity Ward) for sebaceous cyst in left shoulder, and had been allowed to get up when the attack came on.

May 31st.—Patient had a severe fit of coughing in the evening, and soon after pain came on in right side and went on during the night.

June 1st.—No abnormal physical signs in morning, but in the evening tubular breathing and bronchophony could be heard at right base.

2nd.—There is dulness at the right base, and the auscultatory signs have extended. Patient is dyspnœic, face flushed and livid. Pain in right side is intense on coughing and severe at all times.

3rd.—Sputum frothy, muco-purulent, with few masses of rusty sputum. There are a good many moist râles on both sides of chest in front. Dulness and other signs behind extend higher. Rub in right axilla.

4th.—Less pain; good sleep; vomiting twice in night; herpes labialis; sputum frothy. *Physical signs* extend forward on right to anterior axillary border.

6th.—Patient is suffering considerably from diarrhœa. *Physical signs* the same; breathing less difficult.

8th.—Diarrhœa was checked yesterday, but is now bad again. *Signs* of consolidation at the left base.

9th.—Diarrhœa better after starch and opium overnight.

11th.—Diarrhœa again. Severe pain on left side of chest, and the tubular breathing extends round in front below nipple.

12th.—Pain less; resp. 34; less cyanosis; diarrhœa has ceased; rub below left nipple.

15th.—Patient much better; little pain; more cough, sputum frothy.

Dulness now decreased and extending on both sides only up to ninth rib. Tubular breathing less marked, especially on right.

24th.—Still some pain in left axilla with rub.

July 3rd.—No dulness in front. Breathing very harsh.

10th.—Needle withdrew pus from right base, none from left.

11th.—Mr. Howse operated. Little pus came away, but much drained later. Temperature never rose after this.

13th.—Discharge scanty.

25th.—Dulness at left base clearing up rapidly.

August 11th.—Slight dulness at left base.

16th.—Patient went out. Wound quite healed. All dulness at bases has gone.

Summary of Temperature, Pulse, and Respiration.

May 31st—June 4th.—Temperature between 102° and 104°. Pulse between 100 and 140. Respirations between 46 and 68.

June 5th—11th.—Temperature slowly fell till June 11th it was normal. Pulse had become 112, and resp. 36.

June 11th—13th.—Temperature about normal in the morning, and 100° in evening. Pulse 100—120. Resp. 32—48.

June 14th—19th.—Temperature has been between 100° and 101°.

June 20th—July 11th.—Temperature about 99°—100° in the morning, 100·8°—102·2° in the evening. Pulse 86—120. Resp. 28—48.

Case 16 (1891).—Harry F—, æt. 7, admitted May 29th, 1891, under Dr. Goodhart in Philip.

History.—Measles two years ago; frequent subsequent sore throat.

May 29th.—Went to school with sore throat, but was soon sent home. Had nothing to eat or drink except a little water, which he immediately vomited.

Condition on admission.—Drowsy; very dyspnœic (resp. 60); inspiration all over chest loud and harsh; on right side expiration prolonged, inspiration not so harsh as on left; also a râle now and again; resonance good; tactile vocal fremitus normal. *Circulation* normal. *Urine* febrile; no albumen.

June 1st.—Herpes facialis; delirium; refusing food.

2nd.—Patient sleeps sitting up; resonance slightly diminished at right base.

5th.—Patient complains of a little pain in the back; he has had Pulv. Doveri gr. iij o.n., which was omitted yesterday and Quin. Sulph. gr. j 4tis horis substituted.

8th.—Tubular breathing at angle of right scapula and a doubtful rub, certainly a rub at angle of left scapula.

10th.—Dulness and diminished entry of air back and front of right side up to angle of scapula, at which bronchial breathing can be heard.

13th.—*Right base behind,* less dulness; *right and left bases behind,* loss of tactile vocal fremitus; *right apex,* prolonged expiration, dulness extends up to the fourth rib on the right; *left in front,* dulness up to fifth rib;

at both bases behind, loud harsh râles, patches of tubular breathing above dulness.

15th.—*Heart* impulse displaced one inch outwards; dilated veins on left chest.

25th.—Mr. Symonds operated in ninth right space in scapular line, and resected ninth rib; twelve ounces of pus were evacuated.

July 2nd.—Tube worked out and was not returned.

23rd.—No discharge.

August 10th.—Wound healed.

12th.—Patient went out well; right side expands fairly on respiration.

Summary of Temperature, Pulse, and Respiration.

The temperature was very irregular from admission till June 5th, usually high in the evening (104·4° May 31st) and about 101° in the morning. Pulse 130—140. Respiration usually about 50.

June 6th to operation on June 25th, temperature was very regular, 99°—100° in the morning, 102°—103° in the evening. Pulse 120—130. Resp. 48—64.

Case 17 (1891).—William James P—, æt. 18, admitted May 14th, 1891, under Dr. Goodhart in Philip.

Family history.—Father and brother have phthisis.

Previous history.—Has had scarlet fever, measles, and whooping cough, and always has winter cough. In January, 1890, his health seeming to fail he got a berth on board ship, and obtained benefit. Last January he again knocked up, and was sent to a convalescent home. Since then he has been doing light messenger work in London.

Present illness.—A month ago caught cold.

May 2nd.—He was seized with influenza, having bad pains in legs, and headache.

12th.—Was obliged to give up work, and went to a doctor, who diagnosed pneumonia.

Condition on admission (May 14th).—Much pain in chest. Face very dark and congested. Skodaic note at both apices in front. Dulness, bronchial breathing, and bronchophony at right base behind, and the same signs in the scapular region left side. Loud rub at the anterior margin of both these dull areas. *Heart* normal. *Urine* febrile.

16th—*Urine* albuminous.

17th.—Tubular breathing coming further forwards. Sputum rusty Patient very flushed.

18th.—Crisis.

20th.—*Signs:* Dulness more marked at right base. Deficient entry at both bases in front.

25th.—Air enters better at right base. Occasional rub.

28th.—"Certainly more resonance over both lungs posteriorly" (Medical Registrar). Needle put in withdrew nothing.

31st.—*Note by Dr. Goodhart*.—"Resonance decidedly better all over chest back and front, and yet temperature, dry cough, and scanty expectoration

strongly suggest pus. On auscultation there is less tubular breathing on right side and more fine râles from eighth rib down, there being only one small patch of tubular breathing vertically below angle of scupula. On left side pleuritic rub, and breathing more or less tubular as high as eighth rib."

June 1st.—Cough and pain in left side worse; much sweating; sputum less scanty, somewhat purulent.

3rd.—About 6 a.m. patient suddenly woke up with urgent dyspnœa (resp. 60), and feeble and rapid pulse (pulse 148). *Signs:* the tubular breathing is heard all over back and in axillæ, and is much more marked than at any previous time.

25th.—To-day a needle was put in at ninth left space, and pus withdrawn.

26th.—Fifteen ounces of pus evacuated.

July 7th.—Fair resonance on right, back and front; still dull on left.

20th.—Diarrhœa; temp. 98°.

28th.—Increased resonance over left nipple. Patient gets up and walks about.

August 1st.—Tube removed.

7th.—Still a patch of tubular breathing below angle of left scapula. Temp. 98·6°. Wound healed.

20th.—Patient went out. Fair entry of air into left lung.

Summary of Temperature, Pulse, and Respiration.

May 14th—17th.—Temp. 102°—103°, pulse 132—142, resp. 48—60.

18th.—Crisis. Temp. 98·4°, pulse 108, resp. 40.

19th—22nd.—Temperature between 98·4° and 99·8°, pulse 84—108, resp. 32—48.

23rd.—To-day the temperature began to range higher at night, lower in the morning. This increased till the difference became very striking; *e. g.* May 31st, morning temp. 99·2°, evening temp. 104·2°, and this kind of temperature continued till operation on June 26th. During this time the morning respirations ranged from 32 to 70, and the evening from 32 to 76, the pulse varied from 110 to 148.

Case 18 (1891).—William S—, æt. 38, admitted September 10th, 1891, under Dr. Washbourn, in John.

History.—Two months ago had pneumonia, and got well enough to return to work. One week ago, or three weeks after returning to work, he was again seized with pain in right chest, and had to take to bed.

Condition on admission (September 10th).—Skin hot; temp. 103°; resp. 42; pulse 130. Lungs: Tubular breathing and dulness at right base as high as the angle of the scapula and extending to the anterior axillary line, at which a loud rub can be heard. Sputum profuse and pneumonic. Cough severe. Heart normal. Urine 1020, no sugar nor albumen. Bowels constipated.

12th.—Breathing bad during the night, so he was tapped, and six ounces of fluid drawn off from right chest. Sputum profuse, no longer rusty. "Temperature is going down, also pulse and respirations."

14th.—Much better. Temperature keeps down. Cough less troublesome. Dulness at right base not so marked; tubular breathing more so. Some dulness and bronchophony at second right space.

17th.—Tubular breathing gone at right base. Expiration prolonged at apex.

18th.—Some pain in front of chest; linseed poultice applied. Much purulent sputum.

21st.—Tubular breathing and râles at right apex. Dulness much less marked.

23rd.—Air enters badly over right lung behind.

28th.—Entry at right base still deficient. Dulness not clearing up. Needle introduced drew off pus.

29th.—*Operation.*—Ninth rib resected below angle of scapula. Patient vomited pus. Circumscribed cavity found communicating with lung by an aperture size of little finger. Amount of pus evacuated was small.

30th.—Discharge through dressings.

October 1st.—Sputum profuse, muco-purulent and blood-stained.

3rd.—Less expectoration.

5th.—Discharge profuse, mainly blood.

November 3rd.—Patient went out. Wound almost healed.

Case 19.—The full report of this case is unfortunately mislaid, but the following abstract was published in the 'British Medical Journal,' January 30th, 1892:—A young man æt. 18 was admitted into Guy's Hospital under Dr. Hale White in March, 1891, a few hours after the initial rigor of a right-sided pneumonia, the diagnosis of which presented no difficulty. The attack terminated by lysis, the temperature falling gradually on the eighth, ninth, tenth, and eleventh days. On the last of these it reached normal. On the fourteenth day a hectic temperature began, and this continued till his death. There was during all this time pain in the right side, and on the twenty-fourth day a localised empyema was found under the right nipple. As the hectic continued an attempt was made to find another collection of pus in front of the chest, but none could be found, so it was determined to try a day or two later to find pus in the back of the chest, but before this could be done the patient died suddenly. From the position of the localised collection found at the back at the autopsy, there is very little doubt but that it would have been found during life. On about the twentieth day slight jaundice was first noticed, together with bile in the urine. The jaundice gradually deepened, the urine became very dark, and the stools pale. There was a fulness over the left lobe of the liver. On the thirty-fourth day of the disease the patient was sitting up in bed, and he suddenly fell back, became unconscious, and died in about ten minutes.

Autopsy.—Except for a small compressed part at the base the right lung was quite healthy. The pleura was adherent. There was a localised collection of pus between the lungs, ribs, and vertebræ, lying close against the spine. The intestines were œdematous; there was much blood-stained fluid in the abdomen, and a large blood-clot occupied the pelvis and the lateral regions of the abdomen. An aneurysm, the size and shape of a Tangerine

orange, was found on the right branch of the hepatic artery. It lay in the transverse fissure of the liver, and pressed upon the hepatic duct. Its wall was very thin, and it had ruptured at its lower part. There was much ante-mortem clot in it. A similar but smaller aneurysm sprang from the left hepatic artery."

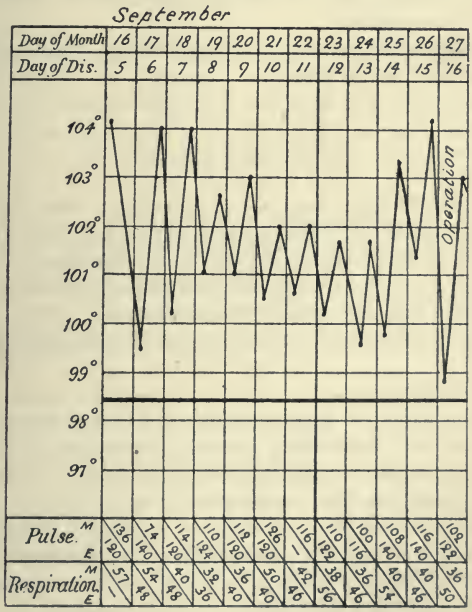
Case 20 (1890).—David H—, æt. 9 years, admitted under Dr. Taylor September 16th, 1890.

History.—Always weakly. Measles two years ago. Influenza nine months ago badly. Three months ago said to have had pleurisy in left side. Three weeks ago was struck in chest (probably unimportant, site of blow forgotten).

September 12th.—Headache, and pain in side; feverishness.

13th.—Doctor diagnosed typhoid; delirious at night; constant vomiting.

14th.—Vomiting ceased about midday. Rigor in afternoon. Pain coming on in right side. Bowels constipated since 12th. No convulsions.



Condition on admission.—Flushed cheeks; shallow respiration (57); anxious expression; dry hot skin; no jaundice; pain severe in right side. *Chest:* tubular breathing and dulness at the right base up to the spine of scapula; dyspnœa worse when he lies on left side. *Circulation:* heart dulness and impulse normal in position.

Treatment.—Ice poultice to right chest.

Progress.—September 17th.—*Right base behind:* dull up to sixth rib,

with well-marked tubular breathing. *Right axilla:* upper part, dull with tubular breathing; lower part, deficient entry; expiration prolonged.

18th.—Coarse râles and rhonchi all round right base. Dulness with tubular breathing and bronchophony up to top of right scapula. Deficient entry in irregular patches. Patient seems worse, is not now delirious. Pulse 128, regular, feeble. Resp. 40.

19th.—Patient much better, has slept well. The signs on the right side extend higher.

20th.—Entry of air worse, fewer râles.

21st.—One or two small patches of tubular breathing and bronchophony over front of left chest. Patch of tubular breathing and bronchophony at right scapular angle.

22nd.—Cough much worse. Voice- and breath-sounds most deficient in right axilla. No cardiac displacement. Coarse râles all over left chest.

23rd.—Four ounces of turbid fluid withdrawn through right eighth space.

25th.—Patient seems better. Temperature rising. *Signs:* much the same; entry of air deficient all over right chest; expiration prolonged over right chest; voice ægophonic.

27th.—*Operation.*—A pint of pus let out of right chest; eighth rib resected. Temperature fell at once.

October 17th.—Tube got blocked. Temperature went up, but fell when properly dressed.

29th.—Patient went to convalescent home; well.

Case 21 (1889).—William L—, æt. 19, admitted under Dr. Wooldridge and subsequently under Dr. Hale White, in John, May 29th, 1889.

History.—Generally healthy. Measles three years ago.

May 27th.—Rigor and sweating this morning; giddiness; later nausea and pain in right chest. Went to bed in afternoon.

28th.—Up in morning, but vomited frequently, and again went to bed in afternoon.

29th.—Pain much worse.

Condition on admission (29th).—Cheeks livid; alæ nasi working; pain great in right axilla. *Respiratory system:* hot dry skin, much dyspnœa; short dry cough, no sputum. Right chest moves badly. Increased tactile vocal fremitus in right axilla. Resonance normal. Medium rattling râles below right nipple and at the base behind. At edge of right lung in front is characteristic fine crepitation. *Circulation:* heart normal; pulse rapid. *Urine:* trace of albumen, chlorides present. Bowels constipated.

Treatment.—Ice poultices. Brandy ʒss 2ndis horis.

30th.—Dr. Wooldridge diagnosed pneumonia. Fluid at right base.

31st.—Condition the same. *Signs:* as before.

June 1st.—Lividity less. Typical rusty sputum yesterday; slight jaundice.

3rd.—Resonance impaired over right lower lobe. Numerous medium inspiratory râles over right lower lobe. Tactile vocal fremitus still increased over right lower lobe. Resonance good above fourth rib in right middle line.

5th.—Patient feels better. Tongue still very foul. Bowels constipated.

7th.—Resonance impaired over right lower lobe behind, good in front. Râles as before and at left lower lobe. Tubular breathing at angle of left scapula. Bronchophony at angle of left scapula.

10th.—Patient feels better. Face still flushed and blue.

12th.—Temp. 104·4°. Bright red rash on cheeks, neck, chest, and arms, also abdomen and loins. Nausea and headache. Ice-bags to axillæ, and sponging.

14th.—Patient still drowsy; pupils act sluggishly. Right chest gives impaired resonance both in front and behind, and even at apex in front. Tubular breathing at angle of right scapula; fine crepitations at angle of right scapula. Still faint rash like rubeola, mostly on chest and abdomen.

15th.—Tubular breathing above dulness on right side.

19th.—Breath-sounds distant at right base; no tubular breathing. Needle put in third space a little external to nipple withdrew pus. *Signs:* Over right base behind and near spine is good resonance; the dulness is confined to right base in front and to right axilla.

20th.—Chest opened in fourth space in mid-axillary line; nine ounces of pus evacuated.

23rd.—Much pus has come away at dressings. Temperature normal.

25th.—Discharge very slight.

July 5th.—Tube removed.

23rd.—Chest resonant, moving well; breath-sounds good.

August 7th.—Patient went out well.

Summary of Temperatures.

May 29th—June 2nd.—Temperature between 102·2° and 103·6°.

June 3rd.—Crisis, with fall to 99·4°.

June 4th—20th.—Temp. 99°—100° in morning, 101°—102° in evening, except June 11th—13th, when it was higher in evening (104·4°).

Case 22 (1888).—Thomas B—, æt. 7, admitted under Dr. Pitt, in John, 10, May 31st, 1888.

History.—Previously healthy.

May 27th.—Sudden pain in back (especially on breathing) on getting up. In bed nearly ever since with pain all over; considerable sweating, free cough; no blood noticed in sputum.

Condition on admission.—Well nourished; skin hot and dry, face flushed, alæ nasi working. Numerous râles in front of chest; dulness, bronchial breathing, and bronchophony over right base behind up to the fifth rib. Cardiac impulse in nipple line, fifth space; no bruits. *Urine:* trace of albumen.

June 2nd.—Still flushed; temperature normal; less dyspnœa. Fine crepitations at right base more numerous. A suspicion of a rub in right axilla.

3rd.—Right chest moving badly; dulness increased upwards a little. Large coarse râles all over. Tubular breathing becomes patchy; less bronchophony. *In front* note impaired below fourth rib in nipple line, with very deficient entry and slight sinking in.

4th.—Dulness complete all over right chest with increased resistance. Tubular breathing over whole of back and front.

5th.—Tubular breathing ill marked at base behind. Many râles below sixth and ninth ribs.

10th.—No entry of air below fourth rib in front, and none at extreme base behind. No dyspnœa.

13th.—Eight ounces of pus evacuated from right chest.

22nd.—Rub in right axilla; no dulness.

23rd.—Wound opened up and rib resected; much bleeding from lung, but no pus.

July 28th.—Tube removed.

August 12th.—Patient went out well.

Summary of Temperature.

On admission (May 31st).—Temp. 104·4°.

Crisis on June 1st; for one morning, of June 2nd, temp. 98°; from this date till operation, June 13th, morning temp. 97°—99°, evening temp. 100°—103°.

June 14th—15th.—Normal or subnormal.

16th—22nd.—Morning temp. 98·4°—99°; evening temp. 100°—102·4°. After second operation on June 23rd it gradually became normal.

Case 23 (1887).—George S—, æt. 27, labourer in brewery, admitted into John, 12, under Dr. Taylor, January 17th, 1887.

History.—January 15th.—Came home from work with diarrhœa and vomiting. One rigor noticed.

Condition on admission.—4 p.m., "evidently very ill." Respiration laboured, 36. Lips congested, and with herpes on them. Skin hot. Temp. 103°. Resp. 36. Pulse 132. Tongue dry, brownish fur down middle. Sputum streaked. *Urine:* Albuminous; sp. gr. 1012. Dulness at right base behind, up to sixth rib; moderate bronchophony; tubular breathing all over dull area; expiration prolonged above. Dulness and tubular breathing extend toward axilla, but do not reach front of chest.

January 18th.—Had a very bad night, getting out of bed. Pulse very small and weak, 108. Skin cold and clammy. The physical signs, which are the same as yesterday, extend up to spine of scapula. A few crepitations about posterior axillary fold. 5 p.m., sudden change for the worse, and death occurred at 6.15 p.m.

Autopsy.—Right chest holds ℥xiij of pus, limited by old adhesions to posterior part of right lower lobe; many old adhesions on left. Right lower lobe in a condition of red hepatisation except for a small portion anteriorly. The other two lobes were only engorged. Two enlarged and caseating bronchial glands, one calcified. *Liver* very large, 128 oz., flabby, distinctly fatty. *Kidneys* large and flabby, structure normal. *Heart* muscle pale.

Case 24 (1887).—Thomas L—, æt. 60, sealskin dresser, admitted John, 8, under Dr. Hale White, June 3rd, 1887.

History.—A long-lived family. No previous history of cough, hæmoptysis, or in fact any illness whatever. Four days ago "caught cold," and has been coughing day and night ever since, bringing up thick yellow stuff, no blood. Pain in chest, difficulty in breathing.

Condition on admission.—Temp. 101·4°. Resp. 64. Pulse 118, small, compressible. Feeble-looking man, much aged. Nose, hands, and feet blue and cold. Lies on right side.

Physical signs.—Left chest: coarse râles and rhonchi all over. Right chest: resonance impaired at apex; total dulness below, where also entry is deficient. Tactile vocal fremitus only slightly impaired; rough pleuritic rub in third space. *Urine:* Trace of albumen.

Treatment.—Right chest aspirated, ʒv of pus withdrawn. Death within a few hours.

Autopsy.—Middle lobe and lower part of upper lobe on right side consolidated. Pleura thickened, ʒiij of pus in right pleural cavity. Arteries thick.

Case 25 (1884).—Mary F—, æt. 26, admitted under Dr. Wilks in Mary, 45, September 2nd, 1881.

History.—Family: A brother died, aged six months, with "enlarged heart," a sister at nineteen of "heart disease and dropsy." *Personal:* Married; since last confinement in 1880 has been attended for cough; four days ago pain in left side on breathing; cough and scanty expectoration ever since. On morning of admission was delivered of a premature child (seven months).

Condition on admission.—Well nourished; anxious expression. Skin moist, not hot (temp. 102°); face flushed; nails clubbed; herpes labialis. *Respiration:* Thoracic, quick, shallow, laboured; no sputum. *Signs:* Left chest moves badly. Tactile vocal fremitus absent in front and left axilla, not well marked on right. Dulness below left fourth rib in front, and about same behind, high-pitched, tubular; pectoriloquy. *Heart* normal. *Urine:* Trace of albumen; chlorides diminished.

September 3rd.—Moist sounds at right base.

4th.—More distress; face blue; more moist sounds, right base, none on left; little cough; severe retching.

5th.—Dulness, tubular breathing, and bronchophony at extreme right base; moist sounds above on left; tubular breathing less loud at base, more so at apex.

6th.—Temperature to-day fell to normal. *Physical signs:* moist sounds have disappeared from right base on left side; on left side dulness as before; tubular breathing more distinct; vocal resonance high-pitched; much less distress; little cough; occasional vomiting.

7th.—On left side dull to spine of scapula behind; breath-sounds almost absent; in front dull up to third rib. On right side no dulness; breathing harsh.

12th.—Signs about same.

14th.—Pulse intermitting. Temperature has begun to rise. It remained fluctuating between 101° and 103° till death.

16th.—Pain and tenderness on left side.

18th.—Cough troublesome during night.

22nd.—Pus drawn off with exploring needle.

24th.—Incision in eighth space; eight and a half pints of pus evacuated.

October 3rd.—Patient rapidly sank and died.

Autopsy.—Aortic valves extensively ulcerated; spleen containing suppurating infarcts; no abscess in lungs.

Case 26 (1883).—Frederick G—, æt. 13, admitted under Dr. Fagge in John, 8, on February 14th, 1883.

History.—Always healthy before this illness.

February 9th.—He had sudden rigors and vomiting, followed by headache and fever.

11th.—Has had much fever; now delirious.

12th.—Pain in right chest and pit of stomach. Bowels well opened with pills, and diarrhœa continued till admission.

Condition on admission (14th).—Face and lips dusky; much drowsiness and irritability. Skin hot and dry. Herpes on cheek and ear.

Respiratory system.—Resp. 48. Alæ working. Respiration mainly thoracic, with interval following inspiration sometimes filled by a groan. Slight cough, scanty sputum, doubtfully rusty. Pain in right chest below nipple. Dulness up to within half an inch of right nipple. Rub about right nipple. Behind, on right, dulness and diminished tactile vocal fremitus below eighth rib. *Heart:* Impulse normal. *Pulse:* 118, feeble. *Urine:* No albumen; slightly alkaline.

16th.—Skin moist. Breathing still catching. Dulness increasing; tubular breathing and a few crepitations over dull area in front. Behind is a pleuritic rub: tubular breathing at angle of scapula.

17th.—Still much pain in right chest. Cough troublesome, dry. Resp. 60. Cyanosis. Loud tubular breathing below right nipple, and numerous coarse consonating crepitations above; a few scattered râles over front of right and left chests. Much distress this evening; exploring needle drew off serum.

18th.—Skin moist; still much distress.

20th.—Better; cough less troublesome; pain gone. Signs as last noted.

22nd.—Sputum scanty, rusty. Tubular breathing less marked in front, very distinct behind.

24th.—Temperature still down; patient cheerful and comfortable. *Signs:* Tubular breathing clearing up.

27th.—More pain again in right chest. Temperature, which had fallen to normal on 19th, has been rising the last two days, and is now 103·6°. Still tubular breathing about right nipple, and a little crepitation. Behind the lung seems to have cleared up.

28th.—Cough troublesome; profuse sweating. Right chest tender; tactile vocal fremitus lost; breath-sounds diminished. Superficial veins enlarged; no œdema, no bulging. Sputum frothy.

March 1st.—Signs on right side, behind: dull up to spine of scapula; tactile vocal fremitus lost. Cardiac impulse displaced outwards. Chest

aspirated; a pint of pus withdrawn. Temperature since February 27th has remained about 103·6°; after aspiration it fell to normal.

6th.—Temperature has been slowly rising the last four days; it is now 103°. Aspirated again; only ʒij of pus withdrawn.

7th.—Incision; no pus found.

14th.—Dulness and tubular breathing up to spine of scapula; well-marked tubular breathing in front. Much discharge of pus to-day from wound.

21st.—Still free discharge from sinus.

April 9th.—Much discharge.

20th.—Discharge scanty. Patient goes into the park when fine.

May 6th.—Patient discharged; sinuses still running.

Summary of Temperature, Pulse, and Respiration.

On admission.—Temp. 104°, pulse 116, resp. 48 and 60 later.

February 15th.—Temp. 105·4°, pulse 136, resp. 68.

16th—19th.—Morning temp. 99°—100°, evening temp. 101°—101·8°, pulse 118—140, resp. 48—68.

20th—22nd.—Temp. 99°—100°, pulse 112—130, resp. 42—48.

23rd and 24th.—Temperature about normal, pulse 100—128, resp. 30—40.

25th—March 1st.—Temperature always higher in the evening, has been rising till yesterday 103·2°, pulse 116—146, resp. 32—48.

1st—7th.—Temperature fell immediately after expiration, but next day began to mount with evening rises till March 5th 103·2°, pulse 92—160, resp. 30—48.

After this the pulse, respiration, and temperature all fell gradually, and became normal on March 15th.

A CASE

OF

MULTIPLE ATROPHYING SARCOMATA OF THE HEAD AND NECK.

By L. A. DUNN.

A. B—, a boy æt. 10 years, residing at Chatham, came under my care at the East London Children's Hospital on September 30th, 1891.

He was a healthy-looking lad, but complained of a sore throat and lumps in his neck.

It appeared that he had been treated at a provincial hospital for sore throat some six months previously, but all attempts to discover the diagnosis made, or the treatment adopted during this time, utterly failed. The mother stated that the lumps in the neck became evident soon after the onset of the soreness of the throat, and have only very gradually increased in size, despite the frequent application of hot linseed-meal poultices.

The parents are both healthy country folk, the mother being thirty-three years of age. She has had eleven pregnancies in all. She first produced a child with a cleft palate, which died young; then followed a miscarriage; then three healthy children, including the patient; then three miscarriages; and, finally, three healthy children. No history of syphilis could be obtained from the mother, and no sign of it could be found in any of the other children.

On admission a firm, slightly nodular mass was found

behind the angle of the jaw on the left side, extending upwards to the parotid gland, and interfering to a slight extent with the movements of the jaw, so that the incisor teeth could be separated for only a distance of about one inch.

It was neither attached to the bone nor the skin, and felt very like the swelling that would be produced by enlargement of the lymphatic glands in this neighbourhood. On the right side of the neck, other smaller isolated swellings were found situated under the anterior border of the sterno-cleido-mastoid muscle, more freely movable than those on the opposite side, and were like them, considered to be enlarged lymphatic glands.

There was some fœtor of the breath, and, when the fauces were inspected, the uvula and greater part of the soft palate were discovered to be missing, the palatal margin being irregular, ulcerated, and coated with a thick layer of yellowish mucus.

As the trouble was thought to arise from syphilis, mercury and iodide of potassium were given internally, whilst the mouth was washed out with permanganate of potash lotion.

On October 14th, a fortnight after admission, a swelling appeared in the substance of the right cheek. It was situated deeply, and quite unattached to the skin and the subjacent bone, rather painful, of a discoid shape, about the size of a florin, and projected on to the mucous aspect of the cheek. It was hard to the touch, somewhat resembling a disc of cartilage, and could be rapped against the neighbouring teeth, giving rise to a very definite click. The temperature, which had previously been normal, arose to 102·4°, and this was accompanied by increase of soreness of the throat and fœtor of the breath, together with rather profuse salivation. The medicine was stopped, and his condition soon improved.

October 30th.—The face is much more swollen on both sides, and the lids of the right eye are very puffy. The masses on both sides of the neck have assumed the same hardness as that in the cheek.

November 7th.—The masses on both sides are gradually increasing in size, and that behind the left ear looks very inflamed, but no fluctuation is perceptible.

12th.—There has been more pain about the swelling in the

right cheek. The skin in this situation is getting involved, and puckers up when the swelling is moved. Oleate of mercury was rubbed into the cheek, but had to be discontinued on account of the pain.

23rd.—The patient complains of pain, and difficulty on swallowing food. The mouth can be opened only just sufficiently wide as to admit of the introduction of the tip of the little finger between the front teeth. A rather large ulcer can be seen, with punched-out edges, on the mucous membrane of the hard palate, opposite the first molar tooth on the left side. The mouth is to be washed out with chlorate of potash lotion, and Decoct. Sarsæ Co. ʒij and Potass. Iodidi gr. ij are to be added to each dose of the medicine he is already taking.

It was now that photograph No. 1 was taken. The swellings have continued to enlarge gradually, and the skin on the left cheek is infiltrated.

There was some apparent benefit from the addition to the medicine, since a note on December 27th states that the boy has been running about the ward for the last week. He seems very well, and all the lumps are decreasing in size. In the right cheek, instead of the rounded swelling there is a depression in the middle of the mass which carries the skin in with it, and looks like an impression which the thumb would leave were it pressed against a mass of soft clay.

On January 2nd, 1892, the report states that there are two masses behind and below the right ear, about the size of walnuts. The upper one has infiltrated the skin, but they are movable independently on the deeper parts, and are separated by a slight sulcus. Occupying the cheek on this side is a hard flat mass about half an inch thick, and reaching from the angle of the mouth to that of the jaw antero-posteriorly, and from the lower margin of the jaw to the level of Stenson's duct in the vertical direction. It is well defined, and projects towards the mucous aspect of the cheek. Over the centre, which is depressed, the skin is smooth, shining, and adherent. The area where the skin is adherent is somewhat circular, with a diameter of an inch and a quarter.

On the left side the induration extends from the anterior border of the masseter muscle to the level of the mastoid process, in the antero-posterior direction, and from the lower

border of the jaw to the zygoma vertically. It is quadrilateral in shape, and well defined both in front and behind. The ear divides the upper half of the mass at the junction of the posterior and middle thirds. The mass is firm to the touch and fairly fixed, and has a deep broad groove in it behind the ear; this tapers upwards, and at the top the skin is drawn up into a small pouch.

The patient had been seen by several excellent observers, three of whom had independently arrived at the conclusion that he was suffering from actinomycosis. It was decided, in consequence, to explore one of the masses; hence on January 16th an incision 2½ inches long was made over the upper of the two swellings on the right side of the neck. Chloroform was the anæsthetic employed. The skin, which was adherent in this situation, was dissected back, and the mass was thus exposed. It was of a greyish-white colour, firm and hard, but scarcely at all vascular. It infiltrated the sterno-cleido-mastoid muscle, as well as the subjacent structures, and had several of the descending cervical nerves coursing through its substance. These were dissected out, but as it was utterly impossible to remove the entire growth, a large portion was excised, the flaps of skin adjusted, and dressings applied. One spot in the posterior flap, where the growth was adherent, remained bloodless. Dr. Washbourn was present at the operation, and made cultivations, but with negative results. A microscopic examination proved the growth to be a sarcoma.

The operation did not cause any trouble, and on February 8th the note states that the wound has quite healed, but the face is greatly swollen, and the mass on the right cheek has reached to the angle of the mouth. The infiltrated skin is smooth, with a pink centre surrounded by a yellowish zone, which shades off into a pinkish-blue margin. The mass on the left side has infiltrated the skin over an area of one inch by one inch and a quarter, which has the same colouring as that on the opposite side. Since November he has lost five pounds in weight.

The patient was exhibited before a meeting of the Clinical Society on February 26th, and was nearly choked whilst partaking of some refreshment. Photographs No. 2 and the

coloured drawing were obtained at this time. After this deglutition became very difficult, and respiration laboured. The growth continued to increase, so that on March 19th the report states that the masses are growing together underneath the chin, and the larynx is almost completely fixed. The eyelids are so swollen that they are completely closed. He has continued to lose weight, since he weighs now only three stone and half a pound, whereas in November he scaled three stone eight pounds and a half. He is so weak that he voluntarily remains in bed, and is breathing with such difficulty that tracheotomy is likely to be required at any moment. Urine sp. gr. 1025, acid, and contains one eighth albumen.

On March 31st, despite all protestations on the part of the resident medical officer, the father took the patient from the hospital down to Chatham, and placed him under the care of Mr. W. H. Smith of that town.

On April 9th, in answer to a letter on the subject, Mr. Smith wrote : " I saw your little patient on Tuesday last, and am of your opinion, that little can be done for him. I fear he cannot hold out very long, as his breathing is so distressed. There has been great œdema round the left eye since he left the hospital. I shall be glad to let you know how he gets on." The next report was kindly furnished by Mr. Smith on June 12th as follows : " You will no doubt be surprised to hear that there is a decided improvement in the little boy ; he is much better in health, and the growth is much smaller in places, and there is much less induration in the tissue surrounding. He swallows and breathes much better, and runs about at play all day. I have subjected him to no treatment whatever."

The writer saw the patient with Mr. Smith on September 21st, and made the following note : " The boy is pale and very emaciated, the skin seeming to be stretched over little else than bone. He breathes with difficulty, there being both inspiratory and expiratory stridor. On the right side of his face the swelling has disappeared, and the right cheek is flattened. The skin in this region is of a yellowish-pink colour, with several tiny blood-vessels ramifying in it, and is peculiarly hard, and leathery to the touch. When the finger

is passed over the smooth surface it is felt to be slightly knobby. This condition is not limited to the cheek, but reaches to the angle of the mouth, invading both the upper and lower lip to a distance of half an inch, and prevents to a great extent the opening of the mouth.

Behind the angle of the jaw on the right side a linear cicatrix is present—the result of the operation,—and although the skin was then implicated, it now appears perfectly natural. No mass or induration can be detected on deep palpation of this region. The growth on the left cheek projects more prominently than when last seen, but is smaller, since it now measures three inches from above downwards, and two and a half inches from before backwards. It is very noticeable, and adheres to the deeper structures. There is a large amount of induration beneath the left angle of the jaw, but the skin here is not invaded.

There is a marked enlargement over the left nasal bone ; it is limited to the superficial aspect of the bone, and does not encroach upon either the orbit or the nasal fossa. All down the front of the neck is a large mass of growth, or rather two of them.

The one, at the upper part and to the right, is about the size of a hen's egg, with the superficial skin completely infiltrated, and appearing of a strikingly red colour owing to the large number of capillary vessels coursing through its substance. The other occupies the remainder of the front of the neck, and tails off as it passes downwards, so that its extremity just overlies the left sterno-clavicular articulation. The skin here is not invaded, but several venules can be seen anastomosing over the surface, a condition which also prevails in the skin over the left clavicle, and the lower lip. The trachea and larynx are entirely obscured, and fixed, by the mass. On the back of the neck is a swelling, one inch by half an inch, having its long axis placed transversely to the upper part of the ligamentum nuchæ. It is in no wise attached to the skin, and is only to a slight extent adherent to the deeper structures. It is impossible to inspect the palate, owing to the difficulty experienced in opening the mouth, but neither fœtor of the breath nor soreness of the throat is complained of.

The skin over the body is harsh and dry. There is no œdema of the extremities. The abdomen is moderately supple, and reveals on palpation nothing suggestive of secondary growths. There is good resonance over the chest on both sides, and the breath-sounds are fairly good, despite the assurances on the part of the mother that the boy is the subject of a severe cough accompanied by a copious mucous expectoration. The present attack of dyspnœa dates some four days back, but has not caused any alarm to the mother, who witnessed a similar attack some weeks ago, from which the patient entirely recovered. The severity of the former attack was such that the noisy respirations could be easily heard in the street below.

There seems to have been one attack of bleeding from the mouth, but this is attributed to a wound of the indurated lip produced by a tooth ; other than this there has been neither ulceration of, nor discharge from, any of the growths. The mother describes the boy's appetite as ravenous, and says he eats and drinks anything, swallowing without the least difficulty. Photographs 4 and 5 were taken on September 22nd.

Both Mr. Smith and the mother remarked upon the variations in the size of the growths ; some would spring up and others almost disappear, and that in the course of but a few days. The patient is very weak, but complains of no pain or tenderness anywhere."

The patient died on October 28th, 1892. The end was sudden. He had been carried down to tea—being too weak to walk,—and ate moderately, appearing to enjoy his food. He was taken back to bed, and was found breathing his last some two hours later.

Between the date of death and the last report nothing very remarkable had occurred, except that the prominent red swellings on the front of the neck had become raw, and bled slightly from time to time. The swellings on the left side of the face had considerably diminished in size, and that extending down the left sterno-mastoid muscle had become more pronounced. The breathing had continued difficult, but the freedom from pain lasted to the end.

A post-mortem examination was made on October 29th.

The body showed much wasting, and rigor mortis was present. There was a prominent swelling rather to the right of the middle line of the neck, the size of an orange, on a level with the thyroid cartilage. The surface was flattened, and covered with a thin dry crust of what appeared to be dried blood, the removal of which exposed a moist surface. When the skin was reflected, a firm mass of whitish growth came into view; it surrounded the trachea, infiltrated the adjacent muscles, and reached downwards as far as the left sterno-clavicular articulation. The walls of the pharynx, too, were attacked by the growth. A slightly enlarged, fleshy gland was found behind the œsophagus, and a second, much larger, behind the root of the left lung. Both lungs were bulky, congested, and somewhat emphysematous. There was no trace of fluid in any of the serous cavities. The heart, liver, spleen, and kidneys were quite healthy. The intestine contained some scybala, but was otherwise natural.

Mr. Targett kindly examined a portion of the growth taken from the front of the larynx, and pronounced it to be a sarcoma. He reports that "The cells are chiefly round, and embedded in a matrix which varies in its character in different parts of the section. In one locality it is finely granular, and the cells are in close apposition; in other parts the matrix is more or less fibrillated, and the appearance is then not unlike richly nucleated fibrous tissue. A few nuclei of the fibrillated matrix can be recognised by their spindle shape, but the section as a whole is quite unlike that of a spindle-celled sarcoma; nor does it resemble a lympho-sarcoma. The microscopic examination suggests that the tumour has originated in loose cellular tissue, and that it possesses a markedly fibrilizing tendency. It is not especially vascular."

The microscopic appearance of that portion of growth removed by operation was almost identical with the above.

Remarks.—The case is of interest first of all from the point of diagnosis. The ulceration of the soft palate, and the masses about the angles of the jaw and cheeks, reminded one forcibly of syphilis, and, indeed, this opinion was maintained by some who were present at the meeting of the

Clinical Society, when the patient was exhibited, in spite of the failure of antisyphilitic remedies to cure the disease, and the microscopic appearance of the portion of growth which had been removed by operation.

Again, the mass in the cheek tallied well with actinomycosis as regards its hardness to the touch, and its tendency to shrink in one part whilst extending in another. The suggestion of actinomycosis was made by three excellent observers independently, but was entirely negatived by the operation. It was certainly unlike an ordinary sarcoma, in that it appeared to spring from many centres, limited to the face and neck, and that it advanced in one part and disappeared in another.

These points are well shown by comparing the accompanying photographs. The disappearance, as a rule, was not quite complete, since a leathery, cicatricial condition of the skin remained. There was, however, an exception to this at the site of the operation, for here, when last seen, the skin was perfectly supple and apparently natural, without a trace of induration in the deeper parts, notwithstanding that at the time of operation the skin was extensively attached, and it was found to be utterly impossible to remove the entire mass. This shrinking of the growth may be explained by its tendency to become converted into fibrous tissue, and may be compared with those atrophic forms of carcinoma met with in the breast.

Mr. Butlin published a somewhat similar case of multiple round-celled sarcomata in a boy, in the 'Transactions of the Pathological Society,' vol. xxix. In this case both testes were attacked, amongst other parts of the body, and after attaining a considerable size, one of them gradually wasted, and became very markedly reduced in bulk.

In conclusion, I have to thank Mr. Targett for reporting on the microscopic appearance of the growth, Mr. H. W. Smith for his kind assistance in following this case, and Mr. W. E. De Korté for the excellent photographs.

DESCRIPTION OF PLATES I AND II,

Illustrating Mr. Dunn's paper on Atrophying Sarcomata of the Head and Neck.

PLATE I.

FIG. 1.—A photograph taken some three months after admission into hospital shows the prominence on the left side of the face, but not the mass which existed on the right side.

FIGS. 2 and 3 were obtained at the same sitting after a portion of the growth had been removed by operation. In fig. 2 the cicatrix can be faintly seen below the ear, whilst the mass infiltrating the skin of the right cheek is very manifest.

FIGS. 3 and 4, obtained one month before death, show the extreme emaciation. In fig. 4 the newly-formed growth in the neck is very marked, the nodule in the middle line behind faintly so. The disappearance of the mass in the right cheek is striking.

In fig. 5 the nodule on the left side of the nose is well shown, in addition to the disappearance of the mass from the right cheek.

PLATE II.

From a painting made shortly before the patient left the hospital. The colouring of the infiltrated skin is excellent, but the circular area on the right cheek appears too much raised; it really was depressed, as can be seen by comparing this Plate with Plate I, figs. 2 and 3.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Danielsson & Co., Collotype.

Dannelgaon & Co, chrome-litn.

ON "PIPERAZIN."

By JOHN FAWCETT, M.D.

This investigation of piperazin was undertaken with the view of determining—

(1) The solvent power it possesses in urine upon uric acid calculi.

(2) The value of it in gout.

(3) The effect of the drug when given to birds, uric acid deposits having been produced in them by injections of potassium chromate.

I. *Its Solvent Action in Urine upon Uric Acid Calculi.*

Methods employed.—(1) Fifteen grains of piperazin were given daily for two weeks to a presumably healthy young adult. The urine during the following twenty-four hours was collected, and allowed to drip over a stone for the next twenty-four, at such a rate that the whole was passed over the stone in that time. The stone was placed in a Soxhlet's fat extractor. This acts automatically, so that when the urine has reached a certain point in the tube it is siphoned off into a receiving vessel below. The whole apparatus was kept in a water-bath, so arranged that the temperature of the urine was that of the body.

The stone was dried at 110° C. and weighed to a constant before and after each experiment.

The urine was kept from decomposing by the addition of a few drops of chloroform.

Weight of stone before experiment, 2·400 grms. At the end of first week 2·404 grms.

Weight of stone before experiment, 2·4435 grms. At the end of second week, 2·6155.

The stone used in the second week, which was a portion of the same one used in the first week, was analysed for me by Mr. Coram, and found to contain 78·5 per cent. of uric acid.

The slight increase in weight was due to a deposit of phosphates, which, on being carefully scraped off, gave the characteristic reactions.

(2) The urine of the same individual as in the above experiment was again collected every day for fourteen days. Two pieces of the same stone were taken. One was placed in the urine, to which fifteen grains of piperazin were added each day, the urine being changed every morning. The other piece was placed in a solution of piperazin in water—fifteen grains to 1000 c.c. of water,—and this again was changed each day.

The two vessels were kept in an incubator at the body temperature.

The stones were dried, and weighed at the end of each week.

i. *Stone suspended in Urine.*

Before immersion.		At end of 7 days.		At end of 14 days.
3·6395 grms.	...	3·6591 grms.	...	3·734 grms.

ii. *Stone suspended in Watery Solution of Piperazin.*

	Before immersion.		At end of 7 days.		At end of 14 days.
1st week ...	3·4795 grms.	...	2·9636 grms.[1]	...	—
2nd week ...	2·8636 grms.	...	—	...	2·484 grms.

[1] The stone had crumbled somewhat, and some few particles may have passed through the fine muslin bag in which it was suspended. The decrease in size, however, was well marked, and very little, if any, had passed through. The bag was opened, a few small particles washed away, and the stone dried and re-weighed for second week's experiment.

Conclusions.—i. After the administration of fifteen grains of piperazin to an individual daily, the urine is useless as a solvent of uric acid stones.

ii. Although a watery solution of piperazin has a marked effect, yet a solution of piperazin in urine in strength used —that is, about 1 in 1000—has no effect whatever.

In the 'Brit. Med. Journal,' June 16th, 1894, Dr. Gordon has described a series of experiments with solutions of piperazin in urine. The strengths of solution of piperazin used were 1, 2, 5, and 7·5 per cent., and no experiments were done with solutions weaker than these.

Now, according to Dr. Gordon's own showing, when 2 grammes of piperazin were given daily, only about 0·3 gramme of unoxidised piperazin was excreted. Taking 1500 c.c. as the normal amount of urine excreted daily, this is only ·02 per cent.

As, therefore, his experiments do not deal with anything like such a percentage of piperazin in the urine, they can scarcely be considered as proving the therapeutical value of piperazin as a solvent of uric acid in the body.

Again, Dr. Gordon has carried out some experiments on powdered uric acid stones the composition of which had been determined.

He gets rid of the phosphates in the urine by first treating with ammonia and then filtering these off. Then he adds 1 per cent. of piperazin to it. This, again, can hardly be considered to be a method of value in testing the therapeutic properties of this drug.

II. *Value of Piperazin in Gout.*

Although this drug in the usual doses is valueless in the treatment of uric acid calculi, yet its great solvent power in a watery medium suggested that the favorable reports of it might result from its power of acting upon uric acid deposits when bathed in a neutral or alkaline medium.

This I proceeded to test by making observations on the urine of the following six cases. All were those of typical chronic gout with uric acid deposits.

In Cases 4 and 6 there was some kidney disease. Case 6

had chronic Bright's disease; and Case 4, when in the hospital under Dr. Moxon in 1884, had been diagnosed as one of "chronic parenchymatous nephritis." Although a slight trace of albumen had been detected in his urine before treatment with piperazin, and also again later, yet while under the treatment there was no albumen in urine. In the other cases the kidneys were apparently healthy.

All the cases were kept on a uniform diet of milk Oiij, fish and two eggs, during the time of treatment, except Case 4, and he was given farinaceous diet with bacon.

The urine was collected for the twenty-four hours, and a specimen from the whole taken for examination. The uric acid was estimated by Hopkins' method, the acid being separated out from the ammonium urate precipitate by means of hydrochloric acid, and titrated against a one-twentieth normal potassium permanganate solution. The urea was estimated by the hypobromite method.

Case 1.—Walter J—, æt. 34.

Previous history.—First attack eleven years before. Big toe-joint only affected. Twelve months later another attack, affecting "big toe-joint, ankles, and feet." For the last eight years has had acute attacks in autumn, and for the last four years a milder attack in the spring. Has drunk "a good quantity of beer."

Present condition (November 13th, 1894).—Numerous tophi in ears. Concretions in all the joints of fingers and toes (phalangeal joints). Deposits in patella and olecranon bursæ. Circulatory system normal. He has now pains in joints of feet and knees, but acute attack has subsided.

November 15th.—Given 15 grains of piperazin daily.

19th.—Given 20 grains of piperazin daily.

20th.—Had acute attack of abdominal pain simulating renal colic. This repeated at intervals during next day. No difference noted in the joints.

Remarks.—The case was a very chronic one. No alteration in amount of uric acid excreted as result of giving piperazin (vide chart).

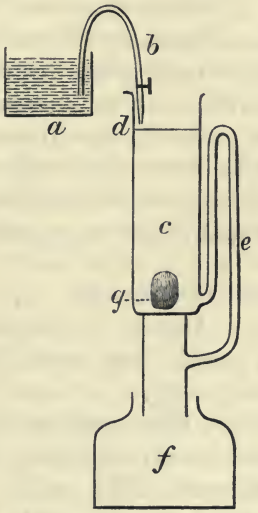


Diagram of Apparatus used in Experiments with Urine upon Stones.

a. Vessel containing urine.
b. Siphon with tap for regulating flow.
c. Soxhlet's extractor.
d. Level at which urine is siphoned off through (*e*) into receiving vessel (*f*) below.
g. Calculus.

Case 2.—Fred. A—, æt. 41.

Previous history.—First attack thirteen years ago. Has had four attacks since, and two of them during this year. Attacks always have commenced in great toe-joints.

Present condition (November 13th, 1894).—The present attack commenced on Thursday, November 8th. Both the metatarso-phalangeal joints are swollen, red, and extremely painful. Attack commenced during night. There are two tophi in left ear. Circulatory system normal.

November 12th.—Piperazin 15 grains given daily.

17th.—Swelling of both joints has disappeared.

21st.—Patient feels quite well again.

Remarks.—The piperazin was given while gout in acute stage, and the pains were apparently much relieved by it. There is, however, no marked result to be noted in the uric acid excreted as result of giving piperazin.

Case 3.—William D—, æt. 55.

Previous history.—Attacks commenced twenty-five years ago; the second attack four years later, and since then has had attacks frequently.

Present condition.—Has swelling of left wrist, and can move shoulders and wrist very little. The joints of fingers and toes are deformed. No tophi. Has had no acute attacks now for about two years, but pains in joints are worse at times than others. Heart normal. Pulse tension rather high.

November 18th.—After taking piperazin 15 grains per diem for four days, he developed acute pain in left wrist and elbow with some swelling.

21st.—The joints are much more swollen, red, and very painful, and skin shiny over them. Severe abdominal pain of paroxysmal character. During last three days the piperazin has been increased to 25 grains. The temperature is raised to 101°—103°.

24th.—Acute pain and swelling of both ankle-joints. Patient put on colchicum and Pot. Bicarb. (November 23rd).

26th.—Pains in joints less, especially the elbows. Swelling still well marked.

Remarks.—Piperazin had no effect upon the pain in joints,

and so the drug was changed. The attack was a very severe one, lasting for some time after discontinuing examination of his urine. Here, with the acute attack, the amount of uric acid excreted was much increased. I do not consider, however, that the piperazin had any effect in producing this.

Case 4.—John D—, æt. 33.

Previous history.—Was in hospital in 1884 under Dr. Moxon, with some œdema of legs and face, and albumen one sixth to one fourth in urine. He was thought to have chronic parenchymatous nephritis. He states he has been quite well since leaving hospital until 1888, when he had an attack of multiple synovitis, and since then has had six or seven similar attacks, the last at the end of 1892. Between attacks has had pains in joints at intervals.

Present condition.—The acute attack for which patient admitted commenced on June 12th, 1894. Many joints affected.

July 12th.—Much deformity of joints, especially of hands and feet. The left shoulder, ankles, left elbow, and first phalangeal joint are the most painful on movement. Tophi in both ears. Heart normal. Systolic bruit at apex has been heard at times since admission. Pulse regular and easily compressible. Urine contains no abnormal constituents, though a slight trace of albumen has been found two or three times previously. No œdema.

July 20th.—Piperazin 20 grains given daily.

23rd to 28th.—Piperazin increased to 30 grains.

21st.—Left knee very painful and tender, and left shoulder hardly movable at all except with great pain.

28th.—Still has pain in joints, but on the whole seems to move joints better. The improvement is only slight.

Remarks.—There was an increase in amount of uric acid excreted during days on which patient was taking 30 grains. The patient thought himself better and more free from pain, and the movements of joints I also thought somewhat improved. Dr. Pye-Smith did not consider the improvement a material one, and he was put back again on colchicum.

Case 5.—Charles C—, æt. 42.

Previous history.—First attack twelve years ago in left big toe-joint. This commenced suddenly during night. From this time till the summer of 1893, when patient was free from an attack for six months, his longest interval of freedom has not been more than three months.

Present condition.—Present attack commenced on November 18th with pain and swelling of right knee. This has now completely subsided. There are gouty deposits in little fingers of both hands, and tophi in both ears (November 28th, 1894).

December 3rd.—Piperazin given in increasing doses from this date till December 15th.

11th.—Had mild acute attack in both little fingers, with some swelling of joints.

18th.—Again a similar attack to that of December 11th.

Remarks.—While fully under influence of the drug, patient developed pain in joints with swelling. There is a slight increase in uric acid excretion to be noticed during the days on which 30 grains were being taken; but as on the following four, when no drug was given, there is also an increase, I think that must be looked upon as a coincidence. The increase, too, is really so small that I do not think any conclusion can be drawn.

Case 6.—John S—, æt. 42.

Previous history.—First attack ten years ago, commencing in right big toe-joint during night. Has had several attacks since.

Present condition.—The present attack began two months ago in right hand, and then attacked the feet. On admission the right elbow, shoulder, and hand were affected. There are tophi in ears and deposits in joints of hands. Patient has "chronic interstitial nephritis," with albumen present, varying in quantity from one twelfth to one sixth.

From December 3rd to 15th patient was given piperazin in doses increasing from 15 to 30 grains per diem.

From 11th to 14th there is an increase in amount of uric acid excreted (vide chart).

15th.—Patient developed a typical acute attack of gout in left elbow-joint.

17th.—Pain remained so severe and swelling so marked, that patient was put upon colchicum and Pot. Bicarb.

Remarks.—Again, while on 30 grains of piperazin there is some increase in amount of uric acid excreted, but as while patient was fully under influence of the drug a very acute attack commenced, the benefit of the piperazin cannot be said to have been great. The pain and swelling also were not relieved by the piperazin, and it was found necessary to put patient on colchicum and Pot. Bicarb., upon which he certainly improved.

CASE 1.—Walter J—. Urine chart.

Date.	Amount in 24 hours.	Sp.gr.	Reaction.	Total urea.	Total uric acid.	Remarks.
	c.c.			gms.	gms.	
Nov. 11	850	1024	Acid	24·6	0·552	Urine normal. No drug given.
12	907·2	1023	„	27·2	0·453	
13	1077·3	1020	„	30·1	0·589	
14	1134	1020	„	30·6	0·449	
15	850·5	1022	Just acid	24·6	0·199	Piperazin 15 grains per diem.
16	1077·3	1022	„	43·0	0·273	
17	850·5	—	—	—	—	
18	1247·4	1022	Just acid	39·9	0·346	
19	1077·3	1020	„	32·3	0·314	Piperazin 20 grains per diem.
20	1020·6	1016	„	24·4	0·278	
21	1340	1020	„	34·8	0·290	
22	1134	1025	„	45·3	0·470	No drug given.
23	1077·3	1020	„	36·6	0·233	
24	1020·6	—	—	—	—	
25	907·2	1022	Just acid	32·6	0·232	
26	1304	1022	„	44·3	0·417	

Case 2.—Fred. A—. Urine chart.

Date.	Amount in 24 hours.	Sp.gr.	Reaction.	Total urea.	Total uric acid.	Remarks.
	c.c.			gms.	gms.	
Nov. 11	453·6	1017	Acid	10·8	0·268	No drug given. Urine normal.
12	907·2	1016	,,	25·4	0·529	Piperazin 15 grains per diem.
13	1190·7	1010	,,	25·0	0·370	
14	1644·3	1012	,,	26·3	0·424	
15	1190·7	1014	,,	23·8	—	
16	1190·7	1016	,,	29·7	0·189	
17	1644·3	—	—	—	—	
18	907·2	1014	Acid	18·1	0·093	Piperazin 20 grains per diem.
19	1134	1014	,,	26·0	0·299	
20	510·3	1014	,,	11·2	0·130	
21	1304	1010	,,	23·4	0·172	
22	1190·7	1016	,,	26·1	0·294	No drug given.
23	963·9	1018	,,	26·0	0·290	
24	907·2	—	—	—	—	
25	1701	1012	Acid	39·1	0·369	
26	1190·7	1016	,,	28·5	0·279	

Case 3.—William D—. Urine chart.

Date.	Amount in 24 hours.	Sp.gr.	Reaction.	Total urea.	Total uric acid.	Remarks.
	c.c.			gms.	gms.	
Nov. 11	1190·7	1013	Acid	23·8	0·482	Urine normal. No drug given.
12	1304	1012	,,	23·4	0·383	
13	1020·6	1018	,,	28·5	0·490	
14	1701	1012	,,	32·3	0·705	
15	1340	1014	,,	28·1	0·592	Piperazin 15 grains per diem.
16	1020·6	1014	,,	19·3	0·278	
17	1190·7	—	—	—	—	
18	1134	1020	Acid	28·3	0·480	Acute pain in left wrist and elbow.
19	1417·5	1010	Neutral	14·1	0·227	Piperazin 20 grains.
20	1701	1020	Acid	44·2	1·123	Piperazin 25 grains.
21	1340	1016	,,	32·1	0·795	Do.
22	1340	1032	,,	77·0	0·925	No drug given.
23	907·2	1030	,,	38·1	0·543	Colchicum and Pot. Bicarb. given.
24	850·5	—	—	—	—	
25	1190·7	1029	Acid	59·4	1·195	
26	1190·7	1029	,,	47·6	0·846	Joints still much swollen, but patient improving.

CASE 4.—John D—. Urine chart.

Date.	Amount in 24 hours.	Sp.gr.	Reaction.	Total urea.	Total uric acid.	Remarks.
	c.c.			gms.	gms.	
July 14	1530	1010	Acid	29·07	0·113	Urine normal. No drug given.
15	1701	1011	„	22·1	0·078	
16	1417·5	1010	„	18·4	0·068	
17	1644·3	1010	„	23·02	0·078	
18	1247·4	1010	„	16·2	0·059	
19	1304	1011	„	18·2	0·052	
20	1672·6	1010	„	21·7	0·083	Piperazin 20 grains per diem.
21	1340	1010	„	21·4	0·061	
22	1247·4	1010	„	19·9	0·063	
23	1190·7	1010	„	16·6	0·071	Piperazin 25 grains.
24	1134	1011	„	14·7	0·293	Piperazin 30 grains per diem.
25	1474·2	1010	„	19·1	0·257	
26	1304	1010	„	15·6	0·078	
27	1530·9	1010	„	19·9	0·283	
28	1247·4	1010	„	14·9	0·230	

CASE 5.—Charles C—. Urine chart.

Date.	Amount in 24 hours.	Sp.gr.	Reaction.	Total urea.	Total uric acid.	Remarks.
	c.c.			gms.	gms.	
Nov. 28	1427·2	1020	Acid	39·7	0·48	Urine pale. No albumen. No drug given.
29	1587·6	1020	„	41·2	0·523	
30	1644·3	1020	„	42·7	0·443	
Dec. 1	1190	—	—	—	—	
2	1020·6	1020	Acid	38·7	0·510	
3	1304	1020	„	36·5	0·451	Piperazin 15 grains per diem.
4	1814·4	1015	„	36·2	0·435	
5	2579·8	1010	Less acid	46·4	0·371	
6	2069·5	1016	„	41·3	0·496	
7	1984·5	1013	„	35·7	0·341	Piperazin 20 grains per diem.
8	1672·6	—	—	—	—	
9	1587·6	1020	Less acid	42·8	0·463	
10	1587·6	1015	„	30·1	0·592	Piperazin 30 grains per diem. (* Accident to precipitate.)
11	2268	1015	Neutral	40·8	0·597	
12	2409·7	1015	Just alk.	33·7	0·532	
13	2466·5	1012	„	34·7	0·495	
14	2268	1012	„	34·0	—*	
15	1871·1	—	—	—	—	
16	2097·9	1010	Just alk.	41·9	0·725	No drug given.
17	1984·5	1010	Acid	34·7	0·496	
18	1927·8	1013	„	36·6	0·520	
19	1956·1	1012	„	31·2	0·412	

CASE 6.—John S—. Urine chart.

Date.	Amount in 24 hours.	Sp.gr.	Reaction.	Total urea.	Total uric acid.	Remarks.
	c.c.			gms.	gms.	
Nov. 28	963·9	1010	Acid	13·3	0·055	
29	1417·5	1010	„	22·6	0·094	Urine pale. Albumen one-twelfth. No drug given.
30	1417·5	1012	„	21·2	0·053	
Dec. 1	—	—	—	—	—	
2	1530·9	1010	Acid	21·4	0·064	
3	1757·7	1010	„	26·3	0·033	
4	2041·2	1010	„	30·6	0·085	Albumen one-twelfth. Piperazin 15 grains per diem.
5	1871·2	1010	„	29·9	0·035	
6	2097·9	1010	„	31·4	0·151	
7	2268	1010	„	31·7	0·031	
8	2211·3	—	—	—	—	Piperazin 20 grains per diem.
9	1984·5	1010	Acid	31·7	0·037	
10	2154·6	1010	„	36·6	0·030	
11	1927·8	1010	„	31·1	0·148	
12	1871·1	1010	„	24·3	0·144	Piperazin 30 grains per diem.
13	2097·9	1010	„	29·3	0·241	
14	1984·5	1010	„	27·7	0·190	
15	1984·5	—	—	—	—	Developed acute attack of gout in left elbow-joint.
16	1927·8	1012	Acid	28·9	0·053	No drug given.
17	1701	1010	„	22·1	0·047	Colchicum and Pot. Bicarb. given.
18	1984·5	1010	„	27·7	0·047	
19	1984·5	1010	„	25·7	0·065	Pain much better and swelling less.

Remarks.

i. In Cases 1 and 2 no increased excretion of uric acid, but a small decrease.

ii. In Cases 3, 4, 5, 6, there is some increase. In 3, however, this was not to be accounted for by the piperazin, but probably by the acute attack, as it continued also after the exhibition of this drug was stopped. In 5 the increase is so slight that I do not think it fair to draw any conclusion. Also the amount passed in the four days after cessation of the drug showed the same amount excreted. In 4 and 6 there is some increase to be noted on giving 30 grains, but not with less.

iii. With the exception of Cases 2 and 4, in the former of which the acute attack was subsiding when treatment began, the pains of the acute stage were not relieved by the piperazin, *i. e.* in Cases 3, 5, and 6. In Case 4 the benefit was rather doubtful.

iv. Cases 2 and 3 developed acute attacks of abdominal pain simulating renal colic. Neither had ever had similar attacks before. I have also heard lately from a very competent clinical observer of a patient, to whom he was giving piperazin daily, having an exactly similar attack.

v. There is no marked alteration in the excretion of urea except in Case 6, and in Case 3 during the acute attack.

vi. There is a small increase in amount of urine passed in Cases 5 and 6.

vii. With regard to the relation of urea and uric acid excretion, it may be noted that on the whole they run parallel. This, however, is not altogether true.

viii. In Cases 4 and 6 the uric acid excretion is extremely small, and in Case 2 the amount is also small. The relation of uric acid to urea is very low in these cases. I intend in a future communication to comment more fully upon both these points.

Thus the results of my observations by no means confirm the favorable reports of many clinical observers as to the use of piperazin in gout.

Sir William Roberts (Croonian Lectures, 1892) states that the addition of piperazin in proportion of 0·1 and 0·2 per cent. to blood-serum or synovial fluid had no effect in increasing the solvent power of these media on sodium biurate, nor did it retard the precipitation of uric acid from serum and synovial fluid.

Stewart ('Therapeutic Gazette,' vol. xvii, and February, 1894) says that he has found it distinctly beneficial, but notes no alteration in urinary constituents. The only change is the diuretic action it exerts.

Levison ('Die harnsäure Diathese,' 1893) states that it is unpleasant and not well tolerated in some cases. In the few estimations he has made he finds no increased excretion of uric acid.

Ebstein and Sprague note that urea and uric acid excretion are not increased.

Biesenthal ('Berlin. klin. Wochensch.,' 1892), on the other hand, has reported cases where the drug seemed very valuable.

Wood ('Therapeutics, Principles and Practice,' 9th ed.) states that sometimes it has a good effect, at others completely fails for no discoverable cause.

Muscular weakness and general depression are said to follow its continuous exhibition. In none of the above cases have I noted any such result.

The drug neither relieves the pain nor does it increase the uric acid elimination. It is also very expensive, and so as it does not appear to possess any greater value, but rather less, than the drugs already recognised in the treatment of gout, I can see no indication for continuing its use further.

III. *Experiments with Pigeons.*

In connection also with this subject I tried to find out some method whereby uric acid deposits could be produced in birds, hoping to be able to test the value of the so-called uric acid solvents in this way.

In referring to previous experiments I found some by Biesenthal and Schmidt ('Berlin. klin. Wochensch.,' 1893). They produced deposits of uric acid in birds by Ebstein's process, *i. e.* by subcutaneous injections of neutral potassium chromate. A one per cent. solution was used.

They found—

i. That deposits were with certainty produced in pigeons as result of injection of ·004 grm.

ii. That in six hens and thirty-nine pigeons treated with the chromate, deposits were produced.

iii. That in sixteen out of twenty pigeons and hens treated with the chromate and piperazin in the form of a pill, no deposits were produced.

iv. That in fourteen out of seventeen pigeons treated in a similar way, no deposits were present.

Experiments on this plan were also performed by Meisels, but I have been unable to obtain access to his original paper.

These experiments I then proceeded to test, but my results, as will be seen from the accompanying tables, are by no means so satisfactory, and seem to show that the method is not reliable. The experiments were performed as follows :

The pigeons were anæsthetised with ether and injected with varying amounts of the neutral chromate.

The punctures were covered with salicylic wool and collodion flexile.

After recovery from the anæsthetic the pigeons apparently experienced no pain as result of the injection.

Thirty experiments were performed (vide table). In fifteen potassium chromate only was injected; in the remainder the chromate was injected and piperazin given in pill.

Results.—i. Chromate injections alone:

In 2 cases	injected with	·01 grm.	... Pigeons died early. No deposits.
3 „	„	·003 grm.	... No deposits produced. Pigeons recovered.
1 case	„	·004 grm.	... Pigeon recovered.
5 cases	„	·006 grm.	... Three died in two days with deposits. Two recovered, and showed no deposits when killed.
4 „	„	·008 grm.	... Two died in two days with deposits. Two recovered, and when killed had no deposits.

ii. Chromate injections and piperazin:

In 2	injected with	·01 grm.	... Died early.
3	„	·003 grm. + 10 grains of piperazin daily	... Two died, one with pathological lesion, the other had deposits. The third recovered, and showed no deposits.
1	„	·004 grm. + 6 grains of piperazin daily	... Recovered. No deposits.
5	„	·006 grm. + 6 grains of piperazin to 3 10 grains of piperazin to 2	All died with deposits in three days.
4	„	·008 grm. + 6 grains of piperazin daily	... Three died in three days, and all showed deposits. One recovered and showed none.

Conclusions.

i. The method is not reliable, for in some cases a certain dose produced the deposits, in others not.

ii. In those pigeons to which piperazin had also been given, deposits of uric acid were still present.

IV. The above method seemed to cause death too quickly,

even if it had been found more reliable. I therefore tried to see if by means of smaller injections at repeated intervals I could obtain the deposits with more certainty, and also without killing the pigeons so soon.

Six experiments were performed.

The pigeons were treated in exactly the same way as before, and apparently experienced no ill effects from the injection, as they took their food well, and showed no abnormal symptoms.

In three pigeons ·001 grm. was injected on alternate days for eight, sixteen, and sixteen injections respectively. On killing the pigeons at end of this time no deposits were found.

In three others ·0005 grm. was injected for five injections, and then ·001 grm. for seven and eight more. On killing these at the end of this period no deposits were found.

V. The deposits produced by Ebstein's process are very beautiful ones. They have the appearance of a coating of fine flour sprinkled over the surfaces of the serous membranes, viz. pleura, pericardium, and peritoneum. The deposit is amorphous, but pure uric acid crystals were obtained from it for me by Mr. Hopkins. The kidneys of the pigeons in which the deposits were present were swollen, and very pale or mottled.

Microscopically the change found is that of acute fatty degeneration and necrosis, the fat in the epithelial cells being well shown by treating with Marchi's fluid.

As a result of the injection the outlet for the uric acid is blocked up, and so the acid is deposited in the tissues.

In conclusion, I owe my best thanks to Dr. Pye-Smith for allowing me the opportunity of examining Case 4 while under treatment, and to Dr. Lloyd of the Lambeth Infirmary for the remaining cases, and for his courtesy throughout. To Dr. Quarry, also of the Lambeth Infirmary, I am much indebted for the great trouble he took in connection with the cases, and in seeing that the treatment was satisfactorily and carefully carried out. I have also to thank my colleague, Mr. Hopkins, for his help in the uric acid estimations I have performed, in the control of some of them, and in many other points connected with this subject.

Table of Experiments.

Injection of Potassium Chromate only.

Date.	Quantity injected.	Result.
Feb. 22.—1	·004 grm.	Recovered.
2	·008 grm.	Recovered. Killed March 3rd. No deposits.
3	·008 grm.	
4, 5	·01 grm.	Died in 12—17 hours. No deposits.
Mar. 6.—1	·006 grm.	Died in 3 days. Deposits.
2	·006 grm.	Do.
3	·006 grm.	Died in 2 days. Deposits.
4	·008 grm.	Died in 28 hours. Deposits.
5	·008 grm.	Died in 2 days. Deposits.
April 3.—1	·003 grm.	All survived. No deposits found when pigeons killed April 11th.
2	·003 grm.	
3	·003 grm.	
4	·006 grm.	
5	·006 grm.	

Injection of Potassium Chromate + Piperazin in Pill by the Mouth.

Date.	Quantity injected.	Amt. piperazin.	Result.
Feb. 22.—6	·004 grm.	6 gr. daily	Recovered.
7, 8	·008 grm.	Do.	Died in 36—48 hours. Deposits well marked.
9, 10	·01 grm.	—	Died in 12—17 hours. No deposits.
Mar. 6.—6	·006 grm.	6 gr. daily	Died in 3 days. Deposits in all.
7	·006 grm.	Do.	
8	·006 grm.	Do.	
9	·008 grm.	Do.	Died in 2½ days. Deposits.
10	·008 grm.	Do.	Recovered, and showed no deposits when killed March 17th.
(All these pigeons were given 6 grains of piperazin on day before injection.)			
April 3.—6	·003 grm.	10 gr. daily	Died in 2 days. Deposits.
7	·003 grm.	Do.	Died.
8	·003 grm.	Do.	Recovered. When killed no deposits.
9	·006 grm.	—	Died in 3 days. Deposits.
10	·006 grm.	—	
(This number were given 4 grains of piperazin on day previous to injection.)			

ON THE TREATMENT OF SUPPURATION OF THE MAXILLARY ANTRUM,

ESPECIALLY WITH REGARD TO THE USE OF A PERMANENT OPENING IN OBSTINATE CASES.

By F. NEWLAND-PEDLEY.

Inflammatory diseases affecting the antrum are of frequent occurrence, and for successful treatment it is essential that they should be recognised at an early stage; especially is this true of inflammatory conditions of the lining membrane. Many of the above are strictly surgical, and I would not venture to discuss them excepting in their relation to the diagnosis of other complaints, but most cases of antral mischief can be traced to the teeth, and the aid of a dental surgeon is generally needed to obtain the best results.

The acute case is generally due to the bursting into the antrum of an alveolar abscess connected with the root of a tooth containing a septic pulp-canal, and the gush of foul pus into the antrum is followed by marked symptoms. Sometimes there is a foul discharge from the nostril noticed in stooping or inclining the head away from the affected side, for in this way the escape of fluid from the antrum into the nose is facilitated. The patient may be conscious of the movement of fluid within the jaw, and there may be constant pain accompanied with tenderness on pressure over the antrum, together with œdema of the cheek. If these symptoms follow an attack of tooth-ache, and a diseased tooth

or roots of teeth can be seen, the diagnosis is pretty clear, and the extraction of the offending tooth will almost certainly be followed by a stream of purulent fluid. If the removal of the tooth leaves a wide opening into the antrum, injections of astringent lotions may complete the cure.

The foregoing description refers to the simplest cases and their most satisfactory termination, which can only be expected when the patient is young and healthy, and when early treatment has cut short the disease. Far different is the usual history of the complaint, which more commonly runs a subacute or chronic course. There may be an utter absence of the prominent physical signs above mentioned; the nasal orifice of the antrum may have become closed, preventing the escape of discharge into the nose, or fluid passing into the nose may pass into the pharynx and not find its exit at the nostril; or it may find its way round at the back of the soft palate and emerge at the nostril of the unaffected side. Both antra may be affected, and the discharge may be unilateral or entirely absent; on the other hand, a discharge from the nostril may be quite independent of antral origin, it may arise from polypi, malignant growths, ozæna, or foreign bodies in the nose. The iritation caused by nasal polypi may set up inflammation within the antrum. Nasal examination will reveal the presence or absence of polypi, growths, and foreign bodies; whilst, as Mr. Christopher Heath correctly observed, in inflammation within the antrum the patient notices a bad smell which is imperceptible to bystanders, but in ozæna the bystanders perceive an evil smell which is imperceptible to the patient.

A sense of fluid surging about within the jaw will be absent when the discharge finds its way into the nose or when the antrum is full, or when the fluid is very small in amount. There may be no tenderness on pressure over the antrum, no œdema, and little deep-seated pain. The pain may be referred to a bad tooth, and antral trouble may not be suspected until a gush of purulent fluid follows the extraction of a tooth, and a probe can be passed up to the floor of the orbit, or until the bulging walls of the antrum confine the diagnosis within a very small compass. When the antrum is distended with fluid the bulging generally affects the anterior

wall, and is best seen, when the mouth is open, at the line where the mucous membrane is reflected from the cheek to the jaw. Later, the swelling may extend as high as the orbit, into which it may burst if unrelieved, or inwards towards the palate, but swelling in the palate is rarely considerable because the anterior wall yields more readily than the palatine.

The character of the bulging aids in distinguishing a solid growth from an abscess or cyst, for solid growths affect the inner and outer plates alike, and thrust the teeth apart. When large they are irregular in outline, devoid of fluctuation, and have a tendency to bleed and break down. In fluid distension of the antrum there is a smooth, rounded swelling generally opposite the anterior wall, and the line of the teeth is normal. In advanced stages the anterior wall disappears and the cyst or abscess has only a membranous covering revealing fluctuation within. On incision a quantity of pus, or cystic fluid containing cholesterin, or a mixture of both, will spurt out, and the finger may be inserted into the antrum.

The frequent absence of pus and the presence of mere cystic fluid suggests that many of these cases are due to the bursting of cysts into the antrum, and it is worthy of note that cysts are common within the lining membrane of the tooth-socket, and are far from rare in the lining membrane of the antrum, where they apparently arise from plugging of the mucous ducts.

The treatment of inflammatory conditions of the lining membrane of the antrum, from whatever cause they may arise, will afford the widest scope for the exercise of judgment, and attention must be directed to the cause of the disease, to the general health of the patient, and especially to the acuteness of the attack. When acute abscess connected with a diseased tooth bursts into the antrum, and the tooth is promptly extracted, an ordinary drainage-tube may be inserted and tied to the nearest tooth. The injection of astringent lotions will be the only remedy needed, and the opening may be allowed to close in two or three weeks. Sometimes the inflammation may be due to a root forced into the antrum in an unsuccessful attempt at extraction, or to food that has found its way through the socket of a

recently-extracted tooth. Such foreign bodies are best removed by enlarging the opening and syringing.

The disease, however, is generally in a subacute or chronic stage when first recognised, and the treatment must extend over weeks or months. The main indication is to drain the antrum thoroughly, and to keep it clean. An opening may be made through the socket of an extracted tooth, or through the anterior wall of the antrum in the canine fossa, or beneath the malar bone. An opening through the socket of a tooth passes through the floor of the antrum, and is very favorable to drainage, but as Mr. Heath pointed out, the opening through the anterior wall of the antrum is less prone to close, is less liable to admit food in mastication, and is very accessible for the use of the syringe. These considerations are of weight, especially when the teeth are perfect, for one would be loth to sacrifice a sound tooth, but as a rule the exciting cause is a decayed tooth, and through its socket the antrum should be opened up. In old patients who have lost the alveolar process or where there has been long-standing inflammation of the socket there will be no difficulty in making the opening, which should be about the size of an ordinary wooden lead-pencil ; but in a young adult with a dense jaw some difficulty will be met. I used to adopt the view that the dental engine should be used for perforating, and I devised various large burrs and drills for this purpose, but the burrs become clogged, and the work can be done quicker and easier with gimlets. Through the perforation thus made the patient should syringe out the antrum, and a pellet of wax or lint should be kept in the aperture to exclude food, and to prevent the gap from healing. If the nasal orifice of the antrum has become closed, forcible syringing must be resorted to, perhaps for some days, before it again becomes pervious, for it is exceedingly important that the nasal orifice shall be open.

Under this treatment the patient may recover, but too often the gum is allowed to heal prematurely, and after a few weeks or months the discharge of pus through a contracted sinus leading into the antrum reveals the sad fact that the antrum is in no better condition than before, and

the disease is becoming chronic. I have seen so many of these failures that in all perforations through the socket of a tooth I insert a metal tube fixed to a small plate or denture. In illustration I might cite a typical case.

Last July I saw, with Mr. Jacobson, a patient from India with a purulent discharge from his antrum. The history extended over two years. The offending tooth had been extracted, the antrum had been syringed through the socket, but the gum had been allowed to heal prematurely, and the result was a narrow sinus over the socket of the extracted tooth through which there oozed a foul discharge. A silver probe could be passed into the antrum. The case had settled down into the usual chronic condition. The patient was a strong, healthy man.

Mr. Jacobson slit up the old sinus and made a large opening into the antrum, chiefly at the expense of the outer alveolar plate. In doing this he made use of a rat's-tail file,[1] which cuts in any direction and does not clog easily. These files cost

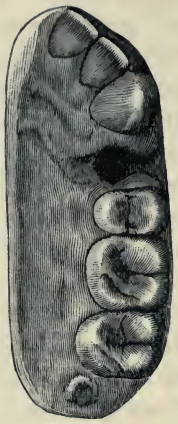
FIG. 1.

Rat's-tail file.

only a few pence, and by breaking off the end of the file as the operation proceeds a fresh portion is brought into play, and rapid progress is made. The antrum was in a very foul condition, and the whole of its interior was thoroughly scraped. A piece of gum-elastic catheter was inserted to keep space for the drainage-tube. After a few days I took a model of the mouth, and repair had taken place so rapidly, that had not the catheter been inserted I should have found great difficulty in adjusting the tube, which in this case had to be more than one inch long. The diameter of the tube was one eighth of an inch, and it was made to fit the nozzle of the syringe recommended. Mr. Jacobson told me that the patient

[1] A most useful instrument, and quite new to me until given me by my colleague (ED.).

Fig. 2.



Model of upper jaw, showing opening into perforation of antrum.

returned to India and the discharge ceased after about three months ; but he was still wearing the tube, and it will be wise to err on the side of caution in deciding when he may finally discard the use of the plate and tube.

There is a special antrum-syringe in which the nozzle is connected to the bulb by means of a piece of rubber tubing about a foot long. Thanks to the flexible tubing, the nozzle can be easily inserted into the drainage-tube, and forcible syringing effected without bending the plate or producing pain. As to the use of holes in the sides of the drainage-tube to facilitate the escape of discharge, if there be much discharge it will easily escape between the tube and the gum.

The tube should be frequently removed for cleansing, and if its replacement be found difficult, a wooden pilot can be fitted to the interior of the tube, to be withdrawn when the plate is in position.

I formerly used a wooden peg to close the tube during mastication, but a patient once forgot the presence of the peg and pushed it into the antrum with the nozzle of the syringe, and much syringing was required to recover the peg. The best stopper for a drainage-tube is a split gold linch-pin surmounted with a head as large as a pea ; the patient can easily grasp this with the fingers in withdrawing the pin. If anything should happen to prevent the plate from being

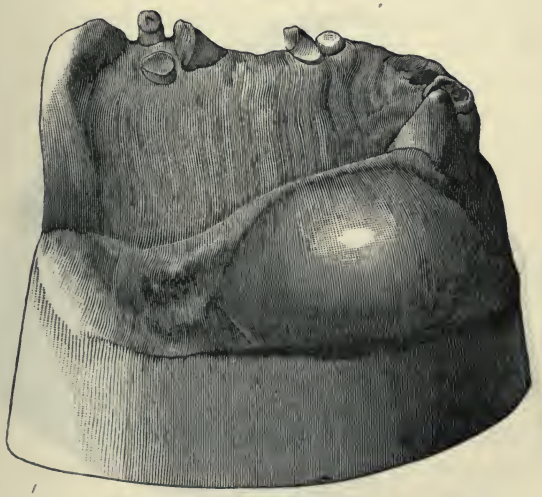
worn for a few days, it may be found necessary to dilate the opening by wearing a piece of gum elastic catheter for a day or two.

The longer the previous history, the longer should the drainage be continued after all symptoms shall have disappeared.

But should we always allow the opening to close? I think not. I can call to mind a few patients who were really chronic cases. They went from one hospital to another; from one dental surgeon to another; they came to me in vain, and I do not know how their cases ended. This determined me to make a permanent opening into the antrum when other means failed.

In 1886 I was asked by Mr. F. V. Mackenzie to see a patient of his, aged about forty, who had an enormously distended antrum which someone had pronounced to be malig-

FIG. 3.

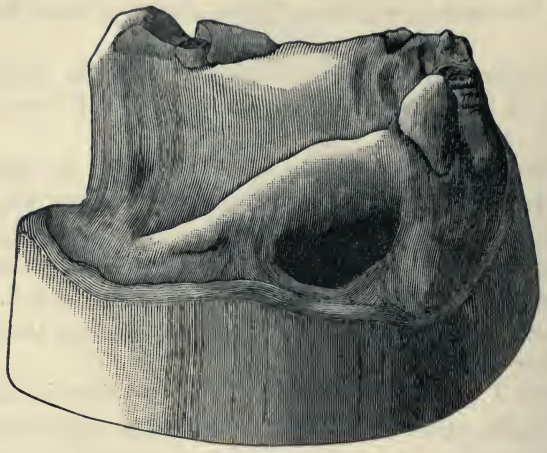


Distended antrum at the time of operation.

nant. It extended up to the orbit, and bulged into the mouth between the cheek and jaw, where it was merely covered by mucous membrane.

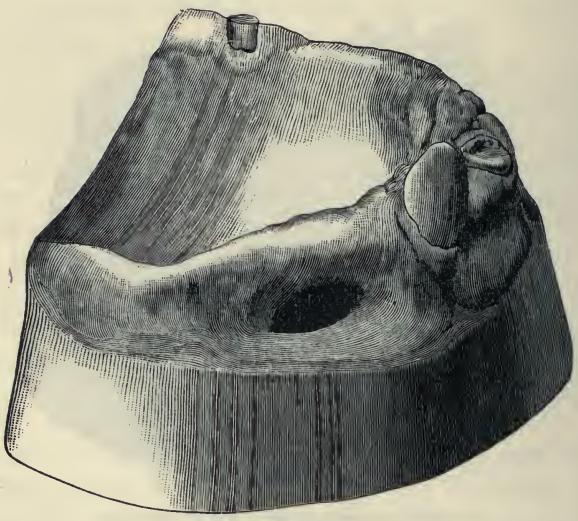
Under nitrous oxide I slit up the cyst and directed the

FIG. 4.



The same three years after operation.

FIG. 5.



The same nine years after operation.

patient to maintain a wide opening by packing the orifice with lint. This he did for several months, and at the end of that time he discontinued all further attention to the antrum, and if food entered it at times he merely rinsed out

his mouth. This showed me how easily a permanent opening into the antrum could be maintained.

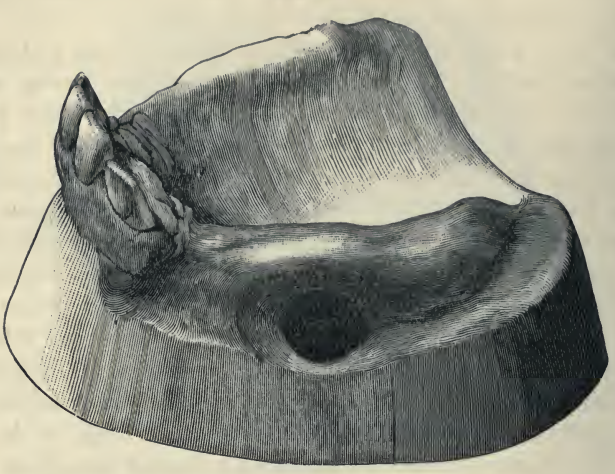
In May, 1892, a dental surgeon sent to Guy's Hospital a patient suffering from inflammation of the lining membrane of the antrum which had existed for two years. During part of that time she attended at a general hospital, where the antrum was syringed through the sinus, and the gum was eventually allowed to close. When I saw her there was a sinus at the summit of the alveolar ridge on the right side in the molar region, from which a purulent discharge issued. Under an anæsthetic I opened up the sinus with a gimlet, and made a plate carrying a metal tube for drainage. The case was thoroughly dressed by Mr. Frederick Tà Bois, a dental student at that time. For months we syringed the antrum with various lotions in turn, for in time one drug seems to lose its effect, and a change does good ; but although we tried Pot. Permang., Hyd. Perchlor., sulphate of zinc, chloride of zinc, iodine water, and whatever else could be suggested, in solutions of varying strength, nothing stopped the discharge from the antrum, and I decided to make a permanent opening because two and a half years had elapsed without improvement. The patient was admitted under Mr. Arbuthnot Lane, who removed the anterior wall of the antrum in December, 1892 ; the contents were merely muco-pus, and the cavity was thoroughly cleared out. The wound showed a strong tendency to heal up, so I fitted a vulcanite denture which prevented the opening into the antrum from closing.

I saw this patient on February 4th, 1895, when she gave me the history of her case, and I took the subjoined model, which shows the present condition.

She is quite sure that even now there is a discharge from the antrum whenever she takes cold. I suggested to her that as she is the subject of post-nasal catarrh, she might be in error as to the source of the discharge, but she is positive that it follows the removal of the plate from the antrum when she is suffering from cold. The mucous membrane of her mouth is very sensitive, and I cannot take a model without previously using a solution of cocaine.

Some of my contemporaries advised me to scrape out the

Fig. 6.

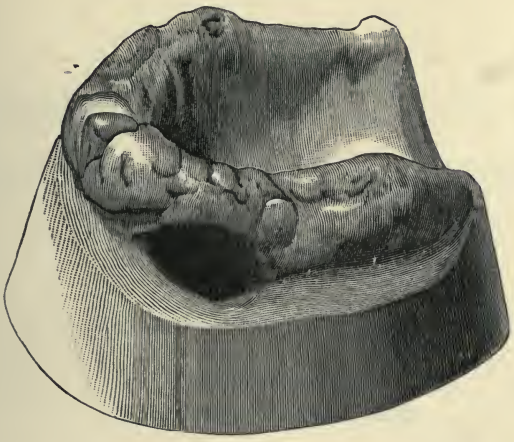


antrum and wash it with a strong solution of chromic acid, allowing it to close after an interval, but I am sure there would have been further trouble in this case. Even if I ignored the periodical discharge from the antrum, the patient's mind had become morbidly centred on her antrum, and every attack of post-nasal catarrh would renew her past anxiety. Now she comes to the hospital from time to time, and is glad to be assured that we can seen into the antrum and there is no cause for anxiety.

On talking over this subject with a dental surgeon of great experience, I was pleased to hear him say that he has a patient whose antrum has a permanent opening. A discharge is noticed occasionally which forcibly suggests that if the antrum were allowed to close, a relapse to the old inflammatory condition would have to be followed by a fresh operation for drainage.

As regards the size of the opening, I do not think the whole of the anterior wall need be removed, and the permanent opening need not be larger than the tip of the little finger. I append the model of a case now under treatment, where a most suitable opening has resulted from the mere incision of a distended cyst or abscess in an edentulous jaw. The history of this case extends over two or three years, and I shall keep the antrum open for several months. If at the

Fig. 7.



end of that time a relapse occurs, I shall establish a permanent opening. There can be no great objection to having an opening into the antrum, provided it be large enough to allow the cavity to be freely rinsed out, and in exceptionally chronic cases where other treatment has failed, it must be a great advantage to abolish the possible retention of inflammatory secretions.

HYPERTROPHY OF THE HEART WITHOUT GROSS ORGANIC LESION.

By THEODORE FISHER, M.D.

(Communicated by the Editors.)

Clinically, hypertrophy of the heart associated with valvular lesions most frequently attracts attention. In the post-mortem room other causes of cardiac enlargement, perhaps overlooked during life, may be discovered, such as adhesion of the pericardium and diseases of the myocardium, of which fibroid degeneration is the most important. Other forms of hypertrophy arising apart from any primary disease of the heart itself may exist in association with disease of the kidneys, or with arterial degeneration. In addition to these cases of large heart due to valvular disease, adherent pericardium, fibroid disease of the heart muscle, hypertrophy consequent upon disease of the kidneys, and obstruction in the arterial circulation, there are examples of enlargement for which no explanation can be found in the post-mortem room. In the carefully-kept records of autopsies in a hospital like Guy's we may sometimes meet with a remark such as the following:—"The case remains obscure, and must be classed as a general cardiac failure with hypertrophy and dilatation, which cases occasionally occur in the post-mortem room."

Overwork has long been recognised as a cause of hyper-

trophy of the heart, and sometimes the pathologist suggests such an explanation when other causes for the enlargement cannot be found. Reference to the clinical report may perhaps show that it affords evidence that lends support to such a view; for example, it may be found that the man was a blacksmith or a stoker, but more often the history points to the cause first pointed out by Bollinger, namely, over-indulgence in alcohol. The liability to sudden cardiac failure in delirium tremens has long been recognised, but it is only within recent years that attention has been drawn to cardiac hypertrophy as a result of alcoholic intemperance. Bollinger, of Munich, noticed that in 1000 necropsies there were 46 cases of death with large heart in which there was no valvular disease, arterio-sclerosis, or granular kidney. He pointed out the excessive beer-drinking of that town. While in the rest of Germany the amount of beer drunk varied from 54 to 186 litres per head annually, in Munich as much as 432 litres were taken. He consequently suggested alcohol as the cause of the hypertrophy.

Such cases of hypertrophy terminating in various degrees of cardiac failure, although overlooked so long, are probably not very uncommon. There have been five cases in the Bristol General Hospital during the past year that may reasonably be considered examples of alcoholic disease of the heart. Three terminated fatally, and upon two of them autopsies were performed. These two cases may be very briefly referred to. One was a rather stout man, aged forty-four, admitted under Dr. Markham Skerritt, October 1st, 1894, with orthopnœa, congested face, and œdema of legs. For fourteen years he had worked in a brewery, and admitted having taken from eight to nine pints of beer a day as well as spirits. For two or three years he had suffered from shortness of breath on exertion, and sometimes would be pulled up when walking with palpitation of the heart and some præcordial pain. The dyspnœa had been worse during the three months that preceded admission. The following notes were made as to the condition of the heart:—"Impulse palpable but cannot be localised, the heart apparently extends outside the nipple line, but the dulness is not increased, possibly owing to emphysema of the lungs." "A marked triple sound"

was audible from a point "1½ inches outside the nipple line in the sixth space, upwards to the third rib, and inwards to the left border of the sternum." There were no murmurs. The pulse was very compressible, but regular and of average size. It was quickened, 120 to the minute. The man died suddenly shortly after he had been sitting up talking to a neighbouring patient. The autopsy revealed a large somewhat dilated heart weighing 17 oz. The dilatation and hypertrophy affected both ventricles equally. The valves were perfectly healthy, and the muscle to the naked eye and microscopically showed no abnormal change. The kidneys were healthy."

The other fatal case was in a woman, aged thirty, sent into the Bristol General Hospital under Mr. C. A. Morton for supposed ovarian tumour. The case proved to be one of cardiac disease with ascites. There was a definite alcoholic history. A systolic murmur was heard at the apex. She died shortly after admission. At the post-mortem examination the heart proved to be dilated and somewhat hypertrophied. It weighed 14½ oz. The aortic valves were slightly thickened, but not deformed. There was no evidence of regurgitation. The other valves were quite healthy. To the naked eye and microscopically the heart muscle showed no abnormal change. The capsules of the kidney were in one or two places slightly adherent to the cortex, but microscopical examination showed the absence of interstitial changes. The liver, although not enlarged, was extremely nutmegged in appearance on section.

As already mentioned, perusal of the Guy's Hospital post-mortem records reveals examples of hypertrophy of the heart associated with an alcoholic history.

The following are brief notes of cases from the records of the years 1884 to 1893. They were under the care of Dr. Frederick Taylor and Dr. Goodhart, who have kindly given me permission to mention them. The first occurred when I was house-physician in 1888.

Year and No. of record.	Sex.	Age.	Occupation.	History of alcohol.	Length of history of cardiac symptoms.	Appearance on admission.	Characters of heart and pulse.	Weight of heart.
1888 No. 200	M.	36	Butcher	"A good deal of beer"	Dyspnœa at night a short time before admission	Very cyanosed; œdema of legs	Impulse feeble; outside nipple systolic apex murmur; pulse weak, slightly irregular, 90	18 oz.
1885 No. 257	M.	49	—	"Rather a heavy drinker"	Œdema of legs 4 months	Extreme dyspnœa; œdema of legs	(No clinical report found)	18 oz.
1893 No. 359	M.	43	Compositor	Drank "his full share of beer"	Two years before there was œdema of legs, which has disappeared; again œdema 10 weeks before admission	Marked dyspnœa; œdema of legs	A triple cantering rhythm; pulse hard	21½ oz.
1889 No. 171	M.	40	Painter	Had drunk freely since 20 years of age, sometimes from 10 to 12 pints a day	Dyspnœa 14 days	Very cyanosed	Impulse 5th space nipple line; soft systolic murmur; pulse small, feeble, regular, 110	16 oz.
1888 No. 107	F.	65	—	"A chronic drunkard"	Œdema of legs 2 weeks	Œdema of legs	Impulse 1 inch external to nipple line; sounds indistinct; no murmurs; pulse at first regular, 90, afterwards irregular	14 oz.
1888 No. 141	F.	49	—	No mention of alcohol, but marked cirrhosis of liver	2½ years before dyspnœa and œdema of legs	Cyanosis and œdema of legs	Soft apex systolic murmur; pulse irregular	16 oz.
1892 No. 252	M.	42	Basket-maker	Had taken too much alcohol for 26 years; lately as much as 1½ pints of whisky daily	Never dyspnœa until 2 days before death	Very cyanosed; œdema of legs	Systolic murmur all over cardiac area	Heart hypertrophied; weight not mentioned.
1889 No. 258	M.	53	Carman	"A heavy drinker"	Dyspnœa 6 months	Œdema of legs	Impulse 6th space; 1st sound reduplicated; pulse regular, 90	22½ oz.

In all of the above cases with one exception the kidneys were quite healthy. In the seventh case on the list the capsules were slightly adherent, but the cortex appeared quite normal. The very marked alcoholic history in this case warrants, I think, inclusion in the list. In the eighth case there was some thickening and contraction of the posterior cusp of the mitral valve, but the pathologist, Dr. Newton Pitt, evidently thought the lesion of no consequence, since he added a note remarking that the cause of hypertrophy was obscure.

A glance at the above brief notes will show that the clinical picture resembles that of other cardiac cases. There is orthopnœa, with cyanosis and œdema of the legs. Perhaps in some cases the cyanosis is unusually well marked, since in no less than three cases it was so great that it was thought advisable to perform venesection.

Examination of the cardiac area may show an impulse displaced outwards, and on auscultation in the majority of instances some abnormal sounds will be heard. At the apex a systolic murmur, due to regurgitation through the mitral orifice that has shared in the dilatation of the left ventricle, is frequently present. This systolic murmur need not, however, be limited to the apex. In one case it was heard over the whole cardiac area, and Dr. Graham Steell,[1] who has published a list of twenty-five cases of alcoholic cardiac failure, frequently mentions a systolic murmur "in all areas." The phrase "in all areas" includes of course a basic systolic murmur. A basic murmur possibly depends upon dilatation of the right side of the heart, mainly in the upward direction, and if the right ventricle should be more dilated than the left, the basic systolic may be the loudest of the murmurs heard. This is illustrated by a case mentioned by Dr. G. A. Gibson[2] of dilated heart due to alcoholic intemperance in a man of twenty-four, and also by a young man aged twenty-eight, at present in the Bristol General Hospital with symptoms of cardiac disease, a dilated heart, and a marked alcoholic history, the only murmur audible being in the pulmonary area. But all varieties of systolic murmur may be absent. In such a case either nothing abnormal may be detected, or a *bruit de galop*

[1] 'Med. Chronicle,' April, 1893, vol. xviii, p. 1.

[2] 'Journal of Pathology,' November, 1894, p. 37.

may be present as in two of the foregoing cases. The pulse appears to be of low tension in the majority of instances; it may be regular or irregular.

The length of time the patients have suffered from symptoms pointing to cardiac failure is of some interest. Bauer[1] speaks of cardiac failure commencing suddenly, and sometimes leading to death in a few days. In one case death was as rapid as this, but in others the history of dyspnœa extended over a period of two years. The history varied in the same way from days to years in Dr. Graham Steell's cases. The average time that dyspnœa had been present was about twelve months before admission.

Before leaving this subject of the effect of alcohol in producing cardiac failure, it may be mentioned that it is a somewhat curious fact that cirrhosis of the liver was the exception rather than the rule. The liver was generally enlarged and nutmeg-like in appearance on section, but in only three instances was cirrhosis present. It was absent even in the man who had been a drunkard for twenty-six years. Apparently the liver may feel the effects of alcohol most in one subject, the nervous system in another, and the heart in a third.

It has already been mentioned that some cases of death from cardiac failure with a large heart may be met with in which alcohol as a cause can be excluded, as well as all the ordinary well-recognised causes of heart hypertrophy. In such cases there may be a history that suggests overwork.

For example, last year a woman, aged forty-eight, died of cardiac disease in the Bristol General Hospital under the care of Dr. Harrison. A large heart was found, free from valvular lesions and unassociated with kidney disease. Here, however, there was no suspicion of alcoholic intemperance. The husband stated that she rarely took more than half a pint of beer a day. She had worked in a glue factory. Her duties were to sort pieces of dried glue, put them into bags, and then to lift these bags into various positions. The husband considered this lifting hard work, and said that it was "the work of a man." The history of the case is somewhat

[1] 'Centrblt. f. innere Med.,' No. 10, 1894; 'Practitioner,' July, 1894, p. 51.

interesting, since attacks of a mild anginal character were present.

Five years before admission, soon after going to bed she was seized with a severe attack of difficulty of breathing, accompanied by dull heavy pain in the cardiac region. The attack lasted about three hours. Since that time she had not been able to exert herself. There was no return of the nocturnal dyspnœa, but from time to time she had sensations of peculiar distress which she said that she could not describe; she felt "as if all the life had gone out" of her. The heart seemed as if it were almost stopped, only a slight flutter being felt occasionally; yet on putting her hand to the chest, pulsations were palpable. From time to time she "would burst out in sweats." Pain was not common, but sometimes the attack commenced with pain "right over the heart." The attack generally lasted about three quarters of an hour. For a year before admission her legs had swelled occasionally, and she had been quite incapacitated from active life. She had not had any serious illness before. She had had three children, two of whom were living. On admission there was orthopnœa, œdema of legs, and marked cyanosis. The heart's impulse was best felt in the sixth space 6½ to 7 inches from the left border of the sternum, but was palpable over the greater part of the cardiac area, and was of a character that on putting the hand over the heart at once suggested the sounds afterwards heard, which were the triple sounds of cantering rhythm. The pulse was regular, 128 to the minute, rather small and compressible. The patient remained in the hospital a considerable time. After being in a month she became extremely cyanosed, with increased œdema of legs and great general distress. Venesection was performed—an operation that seemed to give some relief. During the few days that preceded and followed this venesection she looked as if almost every hour was to be her last; yet slow improvement set in, and four months after admission a note is made stating that she appeared no worse than when first seen. Two months later she died suddenly. As already mentioned, the autopsy showed a large heart free from valvular disease and unassociated with kidney disease. Unfortunately the

weight of the heart has not been recorded, but the organ was kindly kept for me in spirit, and in the fresh state I should think it must have weighed about 24 oz. The coronary arteries were quite healthy, with the exception of one small patch of atheroma at the entrance to the right artery that in no way obstructed the orifice. Microscopic section of the muscle showed nothing abnormal.

Here we have a case that with some reason may be attributed to overwork, and a few of the obscure cases of hypertrophied heart noticed in the Guy's Hospital records may possibly be placed in the same class. In the following three cases the occupations were those of blacksmith, stoker, and labourer in gas-works.

Sex and age.	Occupation.	History.	Clinical aspect.	Characters of heart and pulse.	Weight of heart.
M.[1] 56	Blacksmith	1 year before admission he had dyspnœa and œdema, was laid up 2 months; dyspnœa and œdema returned 4 months before admission	Œdema of legs	Impulse diffused, best marked 5th space internal to nipple line; mitral and tricuspid systolic murmurs; pulse 120, small, irregular	25 oz.
M.[2] 48	Stoker	Dyspnœa for 4 months; œdema of legs 14 days	„	Impulse not seen or felt; sounds normal; pulse at first regular, 105, afterwards irregular	19 oz.
M.[3] 49	Labourer in gas-works	Dyspnœa 7 weeks; œdema of legs 2 weeks	„	Sounds at first normal, afterwards slight systolic murmur	24 oz.

In history and clinical aspect, so far as we are able to judge from so limited a number of cases, hypertrophy of the heart from overwork resembles that due to alcohol. These four cases would seem, however, to indicate that the hypertrophy from overwork is likely to be greater than in cases of cardiac failure due to over-indulgence in alcohol. In two of the cases it will be noticed that in spite of the great hypertrophy of the heart, the sounds were described as normal. Recently there has been in the Bristol General Hospital a man

[1] Year and No. of post-mortem record: 1890, No. 318.
[2] „ „ „ „ 1888, „ 249.
[3] „ „ „ „ 1886, „ 204.

aged fifty-five, who had been a ship's stoker for twenty years, with a large heart, the impulse of which was in the sixth space about an inch outside the nipple line. In spite of the hypertrophy, the faint systolic murmur present was so localised that it was overlooked by both the physician and the house-physician. This feeble character, or absence, of a systolic murmur at the apex is somewhat strange, and seems to indicate that in spite of the failure of the cardiac muscle there may be little dilatation of the mitral orifice.

The cases from the Guy's Hospital records of which brief notes have been given do not exhaust the examples of large heart for which no definite cause could be found. Some additional cases were instances of sudden death in men of whom little or no history was known; but one died in the hospital, and clinically resembled the preceding cases that we have believed due to alcohol and overwork. He was a carpenter aged fifty-four.[1] The occupation does not suggest excessive work, and the clinical report gives no history of alcohol. The liver, however, is described as tough, and it is possible that alcohol may have been the cause of the cardiac hypertrophy.

It has been mentioned that there were some cases of sudden death in which the heart was found to be hypertrophied, but the muscle was apparently free from disease. In one of these instances, occurring in a man aged fifty-three[2] who died suddenly in the surgical out-patient department, the liver is described as tough, a point that suggests the possibility of alcohol as a cause for the heart hypertrophy. In one or two cases we have not, however, even some abnormal condition of the liver to suggest an explanation, and have to content ourselves with the fact that some cases of hypertrophy of the heart of obscure causation may lead to sudden death. One man aged fifty-one,[3] under Mr. Bryant for traumatic stricture of the urethra, complained of sudden pain in the chest, and died in a few minutes. The heart weighed 20 oz. (the clinical report was not found, and consequently the occupation remains un-

[1] Year and No. of post-mortem record: 1886, No. 389.
[2] „ „ „ „ 1884, „ 301.
[3] „ „ „ „ 1884, „ 294.

known). Another man, aged forty-two,[1] was found unconscious by a policeman. The heart weighed 18 oz. In a third case death was very rapid, although not sudden. A young man, aged twenty-five,[2] who gave a history of three days' dyspnœa, died within an hour of admission. Nothing was known of his occupation or habits. The heart was dilated and hypertrophied; it weighed 16 oz. Nothing else abnormal was discovered. In a fourth instance sudden death occurred in a case in which possibly there is no suspicion of alcoholic intemperance or overwork. It was in a medical student aged twenty-four,[3] who had suffered from rheumatic fever, and had been attending Dr. Frederick Taylor for an irregular pulse. He died suddenly while walking to the hospital. The heart weighed 20 oz. In such a case as this it is possible that the attack of rheumatism, instead of affecting the valves or pericardium, may have impaired the nutrition of the heart muscle without producing any obvious organic disease.

Five other cases of large heart remain that belong neither to the category of alcoholic cases nor to those of overwork. Three were attributed by the pathologist to general atheroma of the small arteries. One was in a general servant aged forty-seven,[4] another in a man aged fifty-six,[5] and a third in a man aged fifty-three.[6] In two of these cases there was slight interstitial nephritis; not, however, considered sufficient to account for the cardiac hypertrophy. The two remaining cases were in boys. In one case, in a boy aged seven years,[7] the heart weighed 10½ oz., for which hypertrophy Dr. Carrington could offer no explanation. The mitral valves were thickened but not deformed. Perhaps in this case the thickened valves indicated a previous attack of rheumatism, and, as we have suggested in the case of the medical student, the rheumatism may have impaired the nutrition of the cardiac muscle. The other case was that of a boy, aged five,[8]

[1] Year and No. of post-mortem record: 1892, No. 428.
[2] ,, ,, ,, ,, 1893, ,, 313.
[3] ,, ,, ,, ,, 1884, ,, 6.
[4] ,, ,, ,, ,, 1890, ,, 421.
[5] ,, ,, ,, ,, 1891, ,, 122.
[6] ,, ,, ,, ,, 1887, ,, 107.
[7] ,, ,, ,, ,, 1885, ,, 92.
[8] ,, ,, ,, ,, 1892, ,, 384.

under the care of Dr. Newton Pitt, who three months before admission was struck in the left groin by the handle of a truck, the injury producing an arterio-venous aneurysm between the aorta and vena cava near to their bifurcations. After death the heart was found to weigh 6 oz.—more than twice the normal weight for the age of the child. Possibly the obstruction of the aorta may have been the cause of hypertrophy in this case. Dr. W. T. Howard mentions a case in which obstruction of the abdominal aorta by an ovarian tumour is supposed to have led to hypertrophy of the heart ('Johns Hopkins Hosp. Reports,' 1893, vol. iii, p. 285).

With the exception of one or two cases in which it was considered doubtful whether the amount of kidney disease present could explain the heart hypertrophy, these cases complete the list of cases that I was able to discover during the ten years I have mentioned in which no definite morbid lesion in the heart or kidneys could be found.

Eight cases we have attributed to alcohol, three to overwork, and three were considered due to atheroma of the small arteries. Of eight cases that remain, in four the occupation and habits are unknown, but in two the condition of the liver gives some slight reason to suspect indulgence in alcohol. A further knowledge upon these points might have enabled us to classify some of these cases under the headings of alcohol or overwork.

It has been previously seen that some of these cases of cardiac hypertrophy may terminate in sudden death, even during active life. This being the case, it may be interesting to glance at those cases of sudden death that occurred during the same period from fibroid disease of the myocardium. The frequency with which sudden death occurs in fibroid degeneration of the heart is well recognised, and the records for the ten years we have taken give evidence of the dangerous nature of this disease.[1] In ten cases of sudden death brought into Guy's Hospital, fibroid patches were found in the heart muscle unassociated with valvular lesions. In these ten cases, with one exception, there was some hypertrophy of the heart,

[1] The following are the Nos. of post-mortem records of ten cases of sudden deaths from fibroid disease:—1893, No. 50; 1890, No. 429; 1889, No. 279; 1888, 205, 220?; 1887, 5, 56, 54, 420?; 1884, No. 370?

the weight being generally between 14 oz. and 16 oz. In one, however, the weight was only 11½ oz. All the cases were in men, and of the seven in which the ages were known two were as young as twenty-four and twenty-six, but the remaining five were from fifty to sixty-six years of age. At the time of death they appeared to be following their ordinary callings. One, a fish porter, had been carrying loads, but was talking to a companion when he fell dead. Another, a cabman, was standing by his cab, a third walking across London Bridge, and a fourth looking at the Lord Mayor's Show. A fifth possibly died after some unusual exertion, since he was found dead in a railway carriage.

While there were ten cases of sudden death from fibroid disease of the heart, it is somewhat surprising to find that only three were admitted as clinical cases of heart disease. The best marked example amongst these three was admitted under Dr. Goodhart in 1887, when I was clinical assistant. At the bedside it would no doubt be difficult if not impossible to distinguish such a case as this from one of hypertrophy of the heart in which there was no fibroid change in the myocardium. I think I have read in Osler's book on Medicine, but cannot find the passage, that he has seen cases that answer clinically to the descriptions of fibroid disease of the heart that have proved to be cases of cardiac hypertrophy without fibroid degeneration of the muscle. The case above mentioned occurred in a clerk, aged thirty,[1] who had suffered from dyspnœa for six weeks before admission, and from œdema of the legs for two or three days. The heart's impulse was in the fifth space 2 inches below and 1½ inches outside the nipple line. The heart sounds presented no trace of a murmur. The pulse was small, compressible, very irregular, 84 to the minute. Right hemiplegia, due to embolism of the right middle cerebral artery, occurred two days before death. The autopsy showed a large heart weighing 19½ oz., through the ventricles and septum of which numerous fibroid patches were scattered. The coronary arteries are not mentioned in the post-mortem record, but so far as I remember they were quite healthy. The question of syphilis was unfortunately not inquired for during life, but the

[1] Year and No. of post-mortem record: 1887, No. 60.

patient's brother, with whom he lived, told me that his habits were not those that would expose him to infection. In the two other cases admitted to the hospital as cardiac cases the fibroid patches were not so well marked. One occurred in a man, aged sixty-two,[1] admitted to Guy's six months before with bronchitis and symptoms of cardiac disease. The heart's sounds were at first short and snapping, but afterwards a systolic murmur developed. The pulse was rapid, 160, feeble, and irregular. After death the heart was found to weigh 20 oz. There were some fibroid patches in the septum, and the coronary arteries were slightly atheromatous. The third case was in a man, aged forty,[2] a coachman at hotel stables, who drank from three to four pints of beer a day as well as spirits. Dyspnœa had been present for fourteen weeks. The impulse was diffused, the first sound of the heart short and sharp. The pulse was small but regular, 100 to the minute. At the autopsy the heart weighed 20½ oz. Small fibroid patches occurred along the distribution of the coronary arteries, but the coronary arteries themselves were healthy.

We have, then, three cases of fibroid disease of the heart in which cardiac failure was comparatively gradual, and ten in which it was apparently quite sudden, whereas on the other hand there were twelve cases of hypertrophy of the heart in which the muscle after death appeared healthy that were admitted to Guy's Hospital as cardiac cases, but only four cases of the same disease in which death took place in a few minutes.

One other point may be mentioned in comparing these two forms of affection of the heart. The frequency of cardiac pain in fibroid disease is well known, but possibly it may be present in other cases of heart enlargement. In one of the cases of hypertrophy of the heart probably due to overwork, there was occasional præcordial pain occurring in association with sensations of indescribable distress. In another case of cardiac hypertrophy, sudden death was preceded by complaint of pain. These facts seem to show that we have not only a liability to sudden death in cases of hypertrophy of the heart in which disease of the myo-

[1] Year and No. of post-mortem record: 1892, No. 220.

[2] „ „ „ „ 1893, „ 205.

cardium is absent, but also that cardiac pain may occasionally be present. Possibly, however, the prognosis is less serious than in fibroid disease, and even where pain is present cardiac failure may be gradual rather than sudden. Dr. Balfour[1] mentions that in cases of angina pectoris the less evident the signs of cardiac dilatation, the worse is the prognosis. It is possible that the more serious cases of which he speaks are fibroid disease, and the comparatively hopeful cases are hypertrophy of the heart without gross lesions of the heart muscle. In the suddenly fatal cases of fibroid disease to which we have referred, in no instance the heart weighed more than 16 oz.; while in the other cases of cardiac hypertrophy in which sudden death occurred, no heart weighed less than 18 oz. Here the somewhat curious fact must be mentioned that in the cases of fibroid disease that terminated by slow cardiac failure, the heart weighed from 19½ oz. to 20½ oz.

The consideration of cases of fibroid disease of the heart has led us in a cursory way into the question of diagnosis. It may occur to some that possibly not only fibroid disease but fatty degeneration of the heart may be mistaken for the cases of hypertrophy of the heart that we have been considering. The old ideas as to the frequency of fatty degeneration of the heart have now passed away. Fatty degeneration in varying degrees occurs frequently enough, but rarely as the sole pathological manifestation of a more or less chronic cardiac affection, at least apart from general obesity. In general obesity, however, the heart may suffer with the rest of the body. There were two cases of death from heart failure in stout women, one described as "enormously stout,"[2] the other as "very fat indeed."[3] Their ages were respectively thirty-six and thirty-nine. Another case of death with a large fat-covered heart occurred in a very stout carman, aged about fifty,[4] who fell dead when getting down from his cart. Two other cases [5 and 6] of sudden

[1] 'The Senile Heart,' p. 132.

[2] Year and No. of post-mortem record: 1893, No. 173.

[3] ,, ,, ,, ,, 1888, ,, 410.

[4] ,, ,, ,, ,, 1884, ,, 420?

[5] ,, ,, ,, ,, 1889, ,, 17.

[6] ,, ,, ,, ,, 1891, ,, 84.

death are recorded in men where the heart was loaded with fat, but whether or not there was general obesity is not mentioned. In some of these cases over-indulgence in alcohol may have aided the cardiac failure. There was cirrhosis of the liver in the woman aged thirty-six, and alcoholic intemperance was thought "probable" in the other woman. One of the sudden deaths occurred in a carman, and carmen are perhaps more addicted than most men to an immoderate use of alcohol. In three of the five cases above mentioned we have obesity distinctly mentioned, and in all probability fatty disease of the heart rarely occurs without general excess of adipose tissue. Under these circumstances we shall not have much difficulty in differentiating the disease from other forms of cardiac hypertrophy.

Other cases of death from heart failure occurred where fatty degeneration was present, but possibly the degeneration occurred during the last few days of life and was not the cause of the cardiac enlargement. Thus a man, aged thirty-three,[1] admitted under Mr. Symonds for cellulitis of the arm, suffered from insomnia and dyspnœa. He died, and at the post-mortem examination a heart weighing 19 oz. was discovered, free from valvular lesions. The heart muscle, however, microscopically examined, showed fatty degeneration. In such a case one cannot but think that toxines of the cellulitis present determined fatty degeneration in a previously hypertrophied heart. Another somewhat obscure case occurred under the care of Dr. Moxon. A woman, aged thirty-nine,[2] was admitted with dyspnœa and œdema of the legs, from which she had suffered for eight months. After death the heart was found to weigh 27 oz. There was no valvular disease, but the muscle fibres had undergone advanced degeneration. Here also one cannot think that the fatty degeneration was the cause of the excessive hypertrophy. It seems more probable that the intrinsically weak muscle fibres of a hypertrophied heart degenerated under some exciting cause. As an illustration of the possibility of a hypertrophied heart rapidly failing in this way I may mention the case of a girl, aged twelve,

[1] Year and No. of post-mortem record: 1889, No. 71.

[2] „ „ „ „ 1884, „ 325.

who died in the Bristol General Hospital. She had been in the hospital for pericarditis, and left with a large heart. Six months after she was readmitted with great dyspnœa. Two days before she had been running about, and the weather being frosty, the mother attributed the rapid onset of alarming symptoms to cold. The day after admission she died, three days after the onset of the illness. The heart was found hypertrophied, and the pericardium adherent. Fatty degeneration was present of so extensive a nature that fresh sections of the heart muscle stained with osmic acid turned almost as black as the layer of fat on the surface of the heart. In such cases fatty degeneration cannot be considered the primary disease. The previous attack of pericarditis had injured nutrition, causing hypertrophy of the heart, and this hypertrophied heart under some exciting cause succumbed to fatty degeneration. Fatty degeneration of this nature is a complication rather than a primary disease, and cannot therefore be put in a class by itself and differentiated from various forms of disease causing enlargement of the heart.[1]

There is one group of cases of hypertrophy, however, that needs a passing comment. That group is hypertrophy associated with granular disease of the kidneys.

We are familiar enough with a large heart associated with kidney disease in the post-mortem room, but in the wards it does not often attract attention. This is partly owing to the fact that like other large hearts it may be extensively covered by the lungs, and partly because it often acts well up till the end of life, and even when it fails, does not necessarily give rise to the symptoms of distress present in other forms of cardiac weakness, since instead of ordinary dyspnœa and œdema of the legs, uræmia or bronchitis may be the most obvious feature. It is well known, however, that in those cases of kidney disease in which the clinical aspect is that of heart failure, differentiation from cases of primary heart disease may be difficult, and it must be particularly difficult to distinguish a large heart associated with red granular kidney from the failing

[1] Cases in which fatty degeneration has been associated with obstruction of the coronary arteries due to disease of the aorta have not been referred to.

heart due to over-indulgence in alcohol or over-work. Such a mistake was made on more than one occasion in the cases previously referred to in this paper. The pulse, however, in cases of large heart due to alcohol or overwork, appears to be generally compressible, and the question arises—is the pulse of the failing heart of red granular kidney generally hard? This point was not investigated so thoroughly as I could have wished, but the Guy's Hospital clinical records show that the pulse is frequently described as hard. In other cases it appears to have been compressible. That either pulse may exist there can be no doubt, and tracings could be given from patients dying with a large failing heart associated with interstitial nephritis illustrating this fact. Of recent years, however, much stress has been laid upon arterial degeneration as a cause of cardiac hypertrophy, and the question naturally arises whether those cases in which the pulse was hard were not instances of hypertrophy due to arterio-sclerosis? This affection of the arteries is generally accompanied by interstitial nephritis, and possibly some may consider that many of the cases of large heart in the Guy's Hospital post-mortem records attributed to granular kidney ought to have been considered secondary to arterio-sclerosis. The frequency of this association of granular kidney with arterio-sclerosis is shown by a list of cases of hypertrophy of the heart given by Dr. W. T. Howard, jun.[1] Of a list of sixty-two cases of cardiac hypertrophy considered to be secondary to arterio-sclerosis in only three instances were the kidneys healthy. This illustrates the fact that renal disease and arterial degeneration are intimately associated, but one cannot help thinking that the importance of the arterial degeneration may have been over-estimated. One thing at least is certain, that disease of the kidneys may lead to cardiac hypertrophy apart from arterial degeneration; and Dr. Howard admits the fact, since he mentions eleven cases of large heart associated with disease of the kidneys in which arterial degeneration was absent. Since renal disease can produce cardiac hypertrophy, one would like to have known why in the list of sixty-two cases Dr. Howard considered the evidence to be in so many

[1] 'Johns Hopkins Hosp. Reports,' 1893, vol. iii, p. 294.

instances in favour of the arterial degeneration. Some writers consider that the renal disease is in some cases secondary to the arterial degeneration, while they do not deny that in others the arterial degeneration may be secondary to disease of the kidneys. Such conclusions seem somewhat illogical. If in one case disease of the arteries occurs first, and in another the kidneys are first affected, but arteries or kidneys rarely suffer alone, it is more reasonable to suppose that both diseased conditions are dependent upon the same cause. Whether, however, too great stress has or has not been laid upon the importance of arterio-sclerosis, and whether this arterio-sclerosis generally precedes or follows the disease of the kidneys, there seems to be little doubt that cardiac hypertrophy, arterial degeneration, and renal disease are frequently combined, and we shall not be far wrong in supposing that the latter disease is present in any case of failing heart associated with a hard pulse. It does not follow, however, that when the pulse is compressible we are dealing with a case of hypertrophy due to alcohol or overwork, since although the pulse is of low tension in such cases, it may also be of low tension in some cases of hypertrophy, secondary to interstitial nephritis. In such a case the history of intemperance or the occupation of the patient may aid in arriving at a correct conclusion.

Not only may it be difficult to say whether a case of hypertrophy of the heart is due to alcoholic intemperance or overwork, or whether it is associated with disease of the kidneys, but hypertrophy due to valve disease may need consideration. Since a systolic murmur is not infrequently present in these cases of enlarged heart, the question of mitral regurgitation secondary to valvular disease will probably have to be considered. Here I may mention a fact that may be thought incredible; it is that perusal of the Guy's Hospital post-mortem records seems to show that mitral dilatation from valvular disease as a cause of death can scarcely be said to exist. Excluding those cases of mitral disease in which the aortic valves were also affected, and also cases of adherent pericardium in children, during ten years there were sixty-two deaths from mitral stenosis and not more than five from mitral dilatation;

and in some of those five cases death was probably not due to the affection of the valves, since alcohol or some other cause may have been in operation. For example a man, aged fifty-five, "was brought in from time to time for cardiac failure brought on by drinking bouts." He died of peritonitis following thrombosis of the mesenteric artery. The mitral valves were "rather shrunk," and there had been some regurgitation. In this case alcohol probably had as much to do with heart failure as the valvular lesion. In another case, in a woman aged thirty-eight, there were fibroid patches in the left ventricle in addition to disease of the valve. Possibly the explanation of this rarity of mitral dilatation from valvular disease may be that when the mitral valves become inflamed, the process is progressive and gradually results in stenosis. While, however, nearly every case of mitral disease will prove to be a case of stenosis, it does not follow that in every case a presystolic murmur will be heard. A systolic murmur only may be heard in such cases, even when the orifice is narrowed down to the size of a penholder, and consequently a case of mitral stenosis may be diagnosed as a case of mitral regurgitation due to disease of the valve. It is not necessary to enter fully here into the differentiation of mitral disease from hypertrophy of the heart due to alcohol or overwork. A glance at the lists of the latter cases will, however, show that they occur most often in men and after the age of thirty-five. Mitral disease, on the other hand, is most common in women, and death generally occurs before the age of thirty-five. The history of intemperance or overwork on the one hand, and of chorea or rheumatism on the other, may be of some help, but even these points of age, sex, and history are not sufficient to enable one to make a positive diagnosis. Possibly some physical signs such as the short first sound of mitral stenosis, or the peculiar irregular pulse of that disease, may be present, but cases will occur in which the diagnosis must remain uncertain.

Pathology.—Hypertrophy of the heart due to overwork is possibly simple enough in its etiology. Hypertrophy may be physiological rather than pathological when the organ is called upon to perform a more than customary amount of

work. For example, the heart enlarges during pregnancy. In the hypertrophy of the heart due to overwork, however, the increased number of contractions and force of heart-beats necessary to increase the rapidity of the circulation may not be the only causes of hypertrophy; another factor may be present, namely, resistance in the peripheral circulation secondary to the strong muscular contractions entailed in the performance of work. Slow muscular movements lower the blood-pressure, but those of more rapid character increase it, and the hypertrophy may be partly dependent upon this increase of blood-pressure. One cannot draw conclusions from a limited number of cases, but three of the cases mentioned in this paper, and another case that occurred in the Bristol General Hospital, seem to suggest that hard work associated with heat may be more likely to cause enlargement of the heart than arduous occupations at ordinary temperatures. Two of the men were stokers, one a labourer in gas-works, and a fourth a blacksmith.

It has been suggested that hypertrophy of the heart due to alcoholic intemperance is of very similar nature to that of hypertrophy due to overwork. It has been thought that the amount of work entailed in getting rid of the large amount of fluid taken leads to hypertrophy. One would think such an idea negatived by the fact that the heart does not hypertrophy in diabetes. The question then arises, does alcohol taken habitually in excess lead to increase of pulse tension? Small doses of alcohol in various asthenic heart conditions increase the force of the heart's contractions, and raise the pulse tension; large doses, on the other hand, may paralyse the vaso-motor centre, and lower pulse tension even in the healthy, but I am not aware whether the effect upon the pulse of habitual over-indulgence in alcohol is known.

Although we probably do not know whether high tension plays a part in the production of cardiac hypertrophy in an alcoholic subject, we may suppose that alcoholic excess may impair the nutrition of the heart muscle, and lead to hypertrophy—an hypertrophy which is not an evidence of strength, but of weakness. That it is an evidence of weakness is obvious, since the heart fails, and leads to the

death of the patient. One other example of cardiac hypertrophy may be referred to that seems to me to be due to disordered nutrition,—the hypertrophy associated with adherent pericardium in children following rheumatism or chorea. Simple adhesion of the pericardium, such as is associated with a pleurisy, does not produce hypertrophy, and therefore the enlargement is not due to mechanical interference with the heart's action. Consequently it seems to me that it must be due to disordered nutrition. This disorder of nutrition may possibly arise in one of two ways—either rheumatic pericarditis affects the nerves, which may have trophic functions; or toxines that give rise to the pericarditis so influence the heart muscle, that although it need not show lesions, it becomes intrinsically weak. This weakness is first manifested by hypertrophy, and finally by death. The fact that pericarditis is far more potent in leading to hypertrophy of the heart in children than in adults is in favour of this view, since the growing heart must be more susceptible to nutritional changes. Possibly in rare instances the poison of rheumatism may thus affect the myocardium without any pericarditis being present.

In the cases mentioned in this paper there is one of sudden death in a medical student with a large heart who had suffered from rheumatism. Possibly rheumatism had caused hypertrophy by disordering nutrition. In alcoholic enlargement of the heart we may suppose that alcohol acts in the same way, or that it injuriously affects the metabolism of a great part of the body, and the products of this disordered metabolism are the poisons that act on the heart muscle. Previously it has been supposed that the hypertrophy of overwork is of simple explanation. It may be, however, that it is not quite so simple as first appears. It has been suggested that overwork and heat combined are possibly more potent than overwork alone. Should this be true it is conceivable that the excessive perspiration diminishes the work of the kidneys, and leads at least for a time to the retention of injurious products of metabolism that affect the heart. While such products of metabolism may act on the heart muscle, it is also possible that they may increase arterial tension as well. Extract of supra-renal

capsules raises arterial tension, and if that organic product can produce the result, other organic substances formed within the body may do the same. Were such an explanation correct, the hypertrophy of alcoholic intemperance, overwork, and Bright's disease might all have the same pathology. In all cases the enlargement would be partly due to the action of toxines upon the cardiac muscle, and partly due to raised arterial tension produced by these same toxines.

Treatment.—Treatment of a case of a large failing heart due to alcohol or overwork may be that of an ordinary case of cardiac disease, and it may be mentioned that in spite of the effect of alcohol in producing hypertrophy, Dr. Graham Steell considers alcohol is necessary as a stimulant in such cases while the patient's life is in danger.[1]

Treatment, however, is not limited to the period in which the heart appears to be in danger of a final breakdown; the more hopeful stage occurs earlier. In this paper we have only briefly referred to a few cases in which the nature of the cardiac affection was discovered in the post-mortem room. In all probability, however, many people possess hearts more or less enlarged, the action of which is sufficiently impaired to incapacitate them from sharing in the activity of the majority of their fellows, but at the same time the action, though impaired, may very slowly give indications that life is threatened. It seems to me possible that such cases comprise a considerable number of those that are benefited by the treatment at Nauheim. Excluding disease of the aortic orifice, there can be no doubt that the great majority of cases of morbus cordis occurring after the age of forty are free from any valvular lesion. There is, in most instances of heart disease after that age, hypertrophy—with some dilatation, due to alcoholic intemperance, overwork, arterio-sclerosis, or renal disease. The myocardium shows little or no pathological change, and it is reasonable to suppose that there is some hope of recovery in those cases in which the cause of the hypertrophy need not remain in continuous operation; and even in those in which the cause cannot be removed, the heart may be benefited for a time. We may suppose that the nutrition of the muscle may be improved, and

[1] 'Med. Chronicle,' April, 1893.

that in some way the treatment at Nauheim improves it. Whether the treatment reflexly influences nutrition through the cardiac nerves, or indirectly through the vaso-motor system, is difficult to say. From a few experiments that I have made upon patients with ordinary baths and exercises, it seems that tepid baths may raise the tension of the pulse unless that tension is already high, while hot baths lower it. The slowly conducted exercises also lower pulse tension, even when that tension is due to disease of the kidneys.

Since writing the above remarks upon Nauheim I have paid a visit to the place. Dr. Theodore Schott kindly allowed me to examine several of his patients who had been greatly benefited by the baths and exercises. As thought probable, the most common cardiac affection amongst the successful cases appeared to be hypertrophy without valvular disease. In some instances Dr. Schott suspected over-indulgence in alcohol as the cause of the enlargement. It is interesting to find that Dr. Babcock, of Chicago, who himself has been a patient at Nauheim, believes the treatment by baths and exercises to be especially useful in cases of cardiac hypertrophy in which valvular disease is absent. Whilst he considers rest to be of great value in this class of cases, he thinks that it may be harmful in so-called idiopathic enlargement of the heart ('Journal of American Medical Association,' December 22nd, 1894, p. 940; and 'Medical Press and Circular,' January 2nd, 1895).

In conclusion, I must express my thanks to the present curators of the Museum for permitting me to make such free use of the careful work of past and present pathologists. The more one glances through the valuable post-mortem records, the more one feels how much knowledge they are capable of imparting even to the superficial reader.

THE QUESTION OF AMPUTATION IN SENILE GANGRENE,

BASED ON A STUDY OF FORTY-EIGHT CASES.

By G. BELLINGHAM SMITH.

In 1883 Mr. Jonathan Hutchinson called the attention of the Royal Medical and Chirurgical Society to the advantages of amputating high up in all cases of senile gangrene. In support of his views he gave notes of five cases of amputation through the thigh for gangrene of the foot. He urged that gangrene recurred only in the flaps when the part was removed too near the disease.

Since then many isolated successful cases have been published in the English journals of high amputation for this disease. The relative value, however, of the different methods of treatment adopted is to be measured only by the consideration of a complete series of cases.

This has already been done in Germany by Heidenhain,[1] who collected a series of cases from the clinic of Kuster. The results obtained showed markedly the advisability of high amputations. Kuster commenced with low operations, but was always obliged to re-amputate higher up on account of recurrent gangrene of the wound. He was in this way

[1] 'Deutsch. med. Wochenschrift,' 1891.

(1) *Cases in which no Operation was performed.*

No.	Sex and age.	Condition of large vessels, &c.	Local condition.	Progress.	Result.	Remarks.
(1)	F., 67	Fatty heart	Toe gangrene	Extension	Died	Operation contra-indicated by gangrene threatening in other foot, hands, &c.
(2)	M., 72	Diabetes	Corns; gangrene of toe	Toe separated	Convalescing	No extension to sole of foot or dorsum.
(3)	M., 49	Locomotor ataxy; syphilis	Perforating ulcer; gangrene of hallux	Healing	Convalescing	...
(4)	M., 83	Paralytic stroke; dementia	Chronic ulcer; foot gangrene	...	*In statu quo*	Operation not wished by friends.
(5)	F., 51	Feeble irregular heart; old exophthalmic goitre	Gangrene of dorsum of foot	Gangrenous skin separated and healed over	Recovered	Gangrene of skin, which separated; ulcer 7½ inches long.
(6)	F., 52	Mitral stenosis; clot in aorta and femorals	Gangrene of foot and leg	Extension to thigh, commencing in other foot	Died	Operation here probably hopeless on account of clot in aorta.
(7)	M., 77	...	Toe gangrene	...	*In statu quo*	...
(8)	M., 61	Arterio-sclerosis	Chilblain; toe gangrene	Other foot threatening	Died	Condition too bad to admit of operation.
(9)	F., 62	...	Ulcer of leg; gangrene of foot; foot dropped off	...	*In statu quo*	Spontaneous separation of foot.
(10)	F., 60	D. arterio-sclerosis	Corn; gangrene of toe	Toe separating; onset of coma	Died	Diabetes; coma; no extension to foot of gangrene.
(11)	M., 44	Loss of pulsation in tibials	Threatening gangrene	...	Relieved	...
(12)	F., 76	Mitral disease; arterio-sclerosis	Gangrene of foot	Typhoid condition	Died	Condition too serious for operation.

forced to amputate through or above the knee in every case of gangrene of the lower extremity in which the mortification had extended to the dorsum or sole of the foot.

Herbert Durham and I have already referred to the necessity of amputating well above the necrosed parts in cases of diabetic gangrene of the lower extremities ('G. H. R.,' 1892).

For the purpose of comparing the different methods of treatment adopted, I have collected all the cases of senile and diabetic gangrene admitted into the surgical wards since the year 1879, numbering forty-eight.

They may be classified as regards treatment under the following headings :

(1) Local expectant, antiseptic treatment.

(2) The removal of dead structures, with the cutting through of tendinous and fascial structures.

(3) Amputations close to the gangrenous area (low amputations). High amputations, including—

(4) Those carried out below the knee.

(5) Those performed through or above the knee-joint.

1. *Cases in which no Operation was performed.*

In these cases there was no interference at all with the gangrenous parts. The foot was dressed antiseptically and kept at rest till the necrosed portions separated.

The cases under this heading may be divided into—

(A) Those in which operation is inadvisable on account of the general condition of the patient. Case 1 is an example where general failure of the circulation with gangrene threatening elsewhere was due to a fatty heart.

(B) Those in which as a rule the gangrene is limited to a small extent, generally one or two toes or a patch of skin, and in which separation of the sphacelus is occurring or promises to occur with either slight or no symptoms of general septic poisoning.

In two of these cases the separation was limited to a toe ; in a third to gangrenous skin on the dorsum of the foot.

In Case 9 the entire foot had dropped off above the ankle before admission. Such an extensive separation appears to be of very exceptional occurrence. The result

(2) *Cases in which there was a Removal of Dead Parts and Incision of Abscesses.*

No.	Sex and age.	Condition of vessels, &c.	Local condition.	Treatment.	Result.	Remarks.
(13)	M., 64	G. arterio-sclerosis	Foot gangrene	Removal of dead parts; sloughy tendon cut away; extension	Death	Amputation advised.
(14)	M., 67	D. arterio-sclerosis	Toe gangrene; abscess in sole	Abscess incised; rapid extension of gangrenous inflammation	Death	Amputation advised.
(15)	M., 71	...	Gangrene of hallux; abscess in sole	Abscess incised; spreading gangrene	Death	...
(16)	M., 73	Arterio-sclerosis	Toe gangrene	Three inner toes and slough removed; extension	Death	Amputation not deemed advisable on account of general condition.
(17)	M., 68	D. arterio-sclerosis	Toe gangrene	Gangrenous parts pared with scissors; extension	Death	...
(18)	F., 60	D. arterio-sclerosis	Toe gangrene; abscess on dorsum	Abscess incised; healed; spread of gangrene	Death	...
(19)	M., 75	...	Toe gangrene	End of toe snipped off; head of phalanx removed; extension	Death	...
(28)	M., 54	...	Toe gangrene	Removal of portions of toes; extension; amputation of the leg	Death	...
(29)	M., 71	Paralysis; arterio-sclerosis	Foot gangrene	Separation of gangrenous foot at transverse tarsal joint; ligaments, &c., cut; extension; amputation through upper third of leg	Death	...
(30)	M., 66	...	Toe gangrene	Extension of gangrene to foot; emphysema; incision; rapid extension after this; amputation through leg	Death	Other foot becoming affected.
(46)	M., 46	Arterio-sclerosis	Toe gangrene	Amputation of the toe; incision into sole; extension of gangrene; amputation through thigh	Death	...

was not satisfactory. The patient, as the result apparently of septic poisoning, had passed from a delirious state into one of imbecility, whilst the wound showed but slight signs of healing.

Heidenhain would limit the expectant treatment to cases in which at most two or three toes are affected; demarcation should be waited for and encouraged; but the removal of dead parts with scissors and forceps is not to be recommended. This point will be referred to later.

There were four cases in which the condition was so serious as to preclude all idea of operation (1, 6, 8, 12); all four died.

Of the remainder, one (10) died of what was apparently diabetic coma during the process of separation of the gangrenous toe.

One case alone (5) went out with the wound healed. The remainder left with slowly healing surfaces, liable at any time to reinfection and recurrence of the gangrene.

It may be noted here that the number of cases in which gangrene remained limited to one or two toes is small. In the majority it spread sooner or later to the foot, when the chance of a line of demarcation forming with subsequent successful detachment of the dead tissues is very small.

2. *Cases in which Operation was limited to the Division of Tendons and Fasciæ in the Removal of Dead Parts, or to the Incision of Abscesses.*

There are eleven cases in which there was removal of mortified parts with the cutting through of fascial and tendinous structures, or in which operative interference was limited to incisions into the dorsum or sole of the foot for purulent collections or spreading phlegmon. In every case this was followed sooner or later by extension of the gangrenous process. In three cases amputation through the leg and in one case amputation through the thigh was performed. All the cases in the series died.

This disturbance, however slight, of the parts seems sufficient to open up channels through which infection of the adjoining parts takes place. The cutting through of tendons

(3) *Amputation of Parts with Minimal Portion of Living Tissue.*

No.	Sex and age.	Condition of vessels, &c.	Local condition.	Treatment.	Result.	Remarks.
(20)	F., 71	Glycosuria	Gangrene of hallux	Amputation through metatarso-phalangeal joint; sloughing of edges of wound	Convalescing	Wound showed but little power of repair.
(21)	M., 62	Arterio-sclerosis	Suppurating corn; sloughy ulcer over metatarso-phalangeal joint of great toe	Amputation through metatarsal bone of hallux; gangrene spreading up leg	Death	...
(22)	M., 60	Arterio-sclerosis	Gangrene of toe; two other toes dropped off; recurrence of sloughing	Amputated 1 to 2 years ago; wound unhealed; Syme's amputation; flaps sloughing	Death	...
(23)	M , 58	Arterio-sclerosis	Gangrenous toes; amputation of toe 4 years ago	Amputation of toes; very slow healing	Recovery	Wound healed in 5 months with exception of small spot.
(24)	M., 45	Syphilis	Toe gangrene	Lisfranc's amputation; very slow healing	Recovery	Wound healed in 11 weeks.
(36)	M., 60	Arterio-sclerosis, alcoholic	Toe gangrene; no pulsation in popliteal or tibial	Amputation through transverse tarsal joint; very little bleeding; later in day re-amputation through thigh	Death	Re-amputation through thigh same day.
(46)	M., 46	Arterio-sclerosis, alcoholic	Toe gangrene	Amputation through metatarso-phalangeal joint; extension of gangrene; amputation through thigh	Death	Re-amputation through thigh.
(47)	M., 79	D. arterio-sclerosis	Toe gangrene	Toe amputated 6 weeks before admission; extension of gangrene; amputation through thigh	Death	Re-amputation through thigh.
(48)	M., 72	Alcoholic	Toe gangrene	Right 5th toe amputated 9 days before admission; extension of gangrene; Lisfranc's amputation; extension of gangrene; amputation through thigh; sloughing; further removal of femur	Convalescing	Re-amputation twice, Lisfranc's, and through thigh.

occasionally favours the spread of inflammation along the tendon sheaths.

The advice of Sir James Paget with regard to cases of diabetic gangrene may be applied equally to those occurring as the result of senility: "The less of local interference in the way of picking off bits of slough and the like, the better." Heidenhain is equally strong in his denunciation of this procedure.

3. *Low Amputations.*

There are nine cases in which amputation was performed close to the sphacelated parts.

As a result we have—

1. Extension of gangrene leading to death or requiring re-amputation.

2. A wound which shows but little power of repair or takes months to heal over.

In two cases (23 and 24) healing eventually occurred in five months and eleven weeks respectively. Case 20 showed but little power of repair, and went out with the wound unhealed.

Two cases (21 and 22) died with extension of gangrene, without further operation being performed; and four cases (36, 46, 47, 48) were re-amputated.

In not one case did a low amputation give a good result, the best being Case 24, in which, after Lisfranc's amputation, the wound healed in eleven weeks.

The results obtainable by this method of procedure are very little better than those obtained by the removal of mortified parts, and for these reasons:

1. The blood-supply is defective.

2. However carefully antiseptic precautions are carried out, it is almost impossible to avoid reinfection of the amputation wound.

Mr. Hutchinson ('Med.-Chir. Trans.,' 1884), in recommending amputation through the thigh in these cases, says, "I would abstain as scrupulously in senile gangrene as in frost-bite from any interference with the knife near to the dying part."

As was pointed out by Mr. Hutchinson, it was on account

(4) *Amputation below the Knee.*

No.	Sex and age.	Condition of vessels, &c.	Local condition.	Treatment.	Result.	Remarks.
(25)	M., 54	Radial arteries not hard; general health good	Anterior part of foot removed 2 years ago at Charing Cross for gangrene; slough at extremity	Amputation at seat of election; arteries of limb not noted	Good healing; recovery	Arteries, radial and temporal, good; heart-sounds good.
(26)	M., 57	D. 15 years; perforating ulcer; anæsthesia; lower limbs wasted and cold; cramp; sugar	Perforating ulcer with black slough below 4th and 5th toes; gangrene of great toe; extension to sole of foot	Amputation at seat of election; gangrene of flaps	Died; atheromatous aorta and pulmonary artery	...
(27)	M., 76	Bad pulse; no pulsation in tibials	Gangrene along outer border of foot	Amputation of upper third of leg; remarkably little bleeding; gangrene of flaps	Died; pyæmia; cirrhosis of liver	Very bad condition before operation.
(28)	M., 54	...	Gangrene of the 1st, 2nd, and 3rd toes	Abscess opened on heel and portions of toes removed; extension of gangrene; amputation through middle third of leg; sloughing of flaps; gangrenous abscess of lung incised	Died	Died 3 weeks after amputation of gangrene of lung; sloughing of flaps ceased; bone exposed and necrosed; amputation delayed nearly 3 months.
(29)	M., 71	Radial calcareous; general condition very bad	Foot gangrenous	Dead tissues separated with scissors at transverse tarsal joint; extension; amputation through upper third of leg; popliteal artery calcareous; gangrene of stump	Died; vessels atheromatous	Bad general condition; amputation only after considerable extension of gangrene 5 weeks after admission.
(30)	M., 66	Arteries seem fairly healthy	Toe and foot gangrenous	Incision made; amputation 5 weeks after admission, when gangrene was extending rapidly; amputation 3 inches above ankle; gangrene of flaps	Died	Amputation delayed; low down in leg.

of the low operation that the practice of amputation was almost universally discouraged, sloughing and gangrene of the flaps being the almost constant sequel. At the same time he sounded a note of optimism with regard to high operations which has not been borne out by the experiences of others. "I wish to urge," he says, "that this occurs only when the part is removed too near the disease." As a matter of fact, gangrene recurred in one of his cases after amputation through the thigh.

Although cases of recovery do occur after removal just above the sphacelated portion, healing is very slow, and from the results one seems hardly justified in exposing the patient to the real and great risks that exist of secondary infection of the wound. The vitality of the parts is so small that their power of resistance to invasion by organisms is of the lowest degree, and still more is this the case when the patient is a diabetic.

4. *Amputation through the Leg.*

There are six cases in which amputation was performed through the leg.

In only one of the six cases did healing take place, death occurring with gangrene of the flaps in the remainder.

As causes of failure in this series we have, in the case of No. 26 a history of diabetes of fifteen years; in Case 30 an amputation carried out too close to the gangrenous area, with a speedy recurrence of gangrene due either to re-infection or to a defective supply of blood; and finally delay in operating in the last four cases till the patients' powers were much reduced.

The recurrence of gangrene in the flaps in five of the six cases points to defective blood-supply. It coincides with the experience of Kuster, who amputated in thirteen cases, below the knee. Of these two healed, two died of gangrene, and nine were re-amputated.

5. *Amputation through the Thigh.*

There are eighteen cases in which amputation through the thigh was performed. Of these, eight died.

If diabetic cases are excluded we get a series of fourteen

(5) *Amputation through the Thigh.*

No.	Sex and age.	Condition of vessels, &c.	Local condition.	Treatment.	Result.	Remarks.
(31)	M., 64	Secondary glycosuria	Gangrene of toes extending on to dorsum	Amputation at junction of middle and lower third; healing by first intention	Recovered	Arteries calcareous; lumen in posterior tibial obliterated.
(32)	M., 72	Secondary glycosuria; large prostate; hæmaturia	Foot gangrene spreading	Amputation at junction of middle and lower third; primary union	Recovered	Clot in popliteal artery.
(33)	M., 57	Arteries thick and hard; pulsation in popliteal felt	Foot gangrene spreading	Amputation just above condyles; primary union	Recovered	Calcareous vessels; lumen in popliteal partially obstructed.
(34)	M., 55	...	Gangrene of leg followed knock three weeks ago	Amputation at junction of middle and lower third; primary union	Recovered	Femoral artery very atheromatous.
(35)	M., 69	Traumatic amputation at first phalanx of great toe; toe amputated	Gangrene of flaps extending to foot	Amputation through lower third of thigh; gangrene of flaps; healing in seven months	Recovered	...
(36)	M., 60	No pulsation in tibials; slight in femoral; alcoholic	Toe gangrene; line of demarcation	Amputation at the transverse tarsal joint; very little bleeding from wound, so amputation at the base of great trochanter was performed later in day; gangrene of stump	Death	No pulsation in common femoral after first amputation; femoral artery thrombosed; atheroma of abdominal aorta and iliacs.
(37)	F., 58	Trace of albumen	Foot gangrene; line of demarcation	Stokes-Gritti's amputation; suppuration in wound; slow repair	Recovered	Transferred to Infirmary with wound healing unsatisfactorily.
(38)	M., 54	General condition bad; cystitis	Foot gangrene	Amputation through lower third of femur; gangrene of flaps; portion of femur removed; slow repair	Recovered	Wound healing on discharge about 7 weeks later.
(39)	M., 65	No pulsation below common femoral	Toe and heel gangrene spreading	Amputation through thigh; femoral artery thrombosed; death from exhaustion; wound did well; no gangrene	Death	In spite of thrombosis of femoral artery there was no recurrence of gangrene.

(40)	M., 73	No pulsation in popliteal or tibials; feeble old man	Hallux gangrene spreading to foot	Amputation high in thigh; thrombosis in femoral artery; very little bleeding; wound sloughing; granular kidneys; pneumonia	Death	Bad condition.
(41)	M., 67	D.	Toe gangrene	Amputation through thigh	Death	Diabetic.
(42)	M., 64	D. arterio-sclerosis; no pulsation in popliteal or posterior tibial; very emaciated	Toe gangrene; line of demarcation	Amputation at middle of thigh; femoral artery very calcareous; very little blood lost; coma	Death	Diabetic; death from coma two days after operation.
(43)	F., 67	Aortic bruit; thin and weak; arterio-sclerosis	Gangrene of foot and lower half of leg; line of demarcation	Amputation through thigh; not much hæmorrhage; partial union by primary intention; thrombosis of femoral artery	Recovered	...
(44)	M.	Albumen; arterio-sclerosis; no pulsation in posterior tibial	Gangrene of foot extending	Amputation through lower part of thigh; arteries calcareous	Death	Chronic Bright's disease.
(45)	M., 64	D. 5 years	Pott's fracture; gangrene of leg	Stokes-Gritti's amputation; anterior tibial obliterated below middle of leg; arteries very calcareous; good union	Recovered	Diabetic; good union; marked decrease of sugar after amputation.
(46)	M., 46	Alcoholic; albumen; feeble pulse in tibials	Toe gangrene; amputation of toe; extension of gangrene	Amputation at lower part of thigh; popliteal artery extremely atheromatous; death 3 days later; flaps in good apposition; no gangrene	Death	Alcoholic; flaps doing well; fatty viscera, &c.
(47)	M., 79	D. arterio-sclerosis	Toe gangrene extending to foot	Amputation through middle of thigh; vessels thickened; death somewhat suddenly about 12 hours after	Death	Diabetic; death from (?) shock.
(48)	M., 72	Alcoholic; perforating ulcer; paralytic stroke, &c.	Gangrene of toes and foot; line of demarcation; toe amputated 9 days before admission; extension of gangrene	Lisfranc's amputation; extension of gangrene; amputation through thigh; sloughing of flaps; further portion of femur removed; discharged with sinuses	Recovered	Failure of low operations; alcoholic; paralytic stroke 4 years before.

cases of operation with five deaths; and this, considering the serious general condition of many of the patients, is a result that one cannot help regarding as exceedingly good. It compares most favorably with the previous methods of treatment.

In the eighteen cases in which amputation was performed above the knee there was primary union in four cases (the four most recent cases). In two others there was good union.

In six cases there was either gangrene or sloughing of the flaps or suppuration in the wound before recovery.

Of the eight cases that died three were diabetic. One died apparently of coma; a second died rather suddenly within twelve hours of operation; there are no details given about the third.

Case 36 suffered two amputations in one day. Amputation was first performed through the transverse tarsal joint for gangrene of the fifth toe. On removal of the tourniquet it was found that there was scarcely any bleeding from the surface of the wound. As no pulsation in the arteries could be felt as high as the common femoral, the leg was later on in the day removed in the upper part of the thigh. In spite of the high amputation gangrene recurred in the stump, and the patient died three days later. The femoral artery was found thrombosed.

In two other cases in which the femoral artery was found thrombosed at the time of operation, death occurred (39, 40). In one of the cases, however, in spite of the thrombosis, the wound was doing well, and showed no signs of inflammation or gangrene up to the time of death, which occurred six days later.

The fourth case (43) in which the femoral artery was thrombosed recovered with partial union by primary intention.

In two cases, 32 and 33, the lumen of the popliteal was encroached on; in one case by clot, in the other by the projection into the interior of rough calcareous plates.

In all the cases but four some note is made about the vessels, which were thickened, atheromatous, and calcareous. In not one case is it noted that the vessels were healthy.

The frequency of extreme calcareous degeneration of the femoral and popliteal arteries, and of their occlusion, is a very strong argument in favour of high amputation. In no case was there any trouble with secondary hæmorrhage.

In four cases was re-amputation performed—35, 36, 46, 48. In three cases a toe was amputated with extension of gangrene necessitating higher operation. In one case Lisfranc's amputation succeeded the removal of a toe, and later on amputation of the thigh was necessitated by recurrence of gangrene. In a fourth case amputation at the transverse tarsal joint was followed by amputation through the thigh.

It is interesting to note in Case 45 the marked diminution of sugar that took place after, and apparently as the result of the removal of the gangrenous tissues. The patient had been under treatment for diabetes for five years. The wound healed well.

The fact that the only cases in which primary union occurred were those in which amputation was performed above the knee, and the extreme frequency of recurrence of gangrene in the flaps in amputation below the knee, point to the conclusion that the nutrition is better above the knee-joint than below it.

Etiology.—The factors involved in the production of gangrene of the lower extremities may be tabulated as follows.

1. Arterio-sclerosis and other diseased conditions of the circulation, leading to an imperfect supply of blood.

2. Supply of blood which is impure, as in Bright's disease and diabetes; this condition is frequently associated with the first factor.

3. Inflammatory disturbance of the local circulation due to—

(*a*) Injury. This in itself may be very trivial, but when associated with the previous conditions is sufficient to cause stasis of the blood-flow.

(*b*) Septic infection; either taking place through a recent wound, such as a cut corn, or originating in some pre-existent condition, such as an ingrowing toe-nail or perforating ulcer.

4. Nerve lesions affecting the nutrition of the tissues, such as neuritis.

The most important factors are undoubtedly the various

degenerative and inflammatory conditions of the arterial walls, atheroma, endarteritis, thrombosis.

The more carefully examinations are made, the more frequently are these lesions found. Of late years, Heidenhain says, every limb amputated in Kuster's clinic has been examined, with the result that as a rule an old thrombus was found occluding one of the arteries of the lower limb, most frequently the popliteal.

In association with the various arterial conditions causing obstruction to the blood-flow will be found in some cases a degenerated and feeble heart associated with atheroma of the coronary arteries.

If we may judge by the present series of cases, gangrene due to debility of the heart without obstructive lesions of the arteries is rare.

Case 1 seems to represent this condition. An old woman of sixty-seven, with a radial that was apparently not atheromatous and with pulsation in both anterior and posterior tibial arteries, developed spontaneously gangrene in the left foot. Before death, gangrene was threatening in the other foot, and in both hands and elbows. The patient was found after death to have a fatty heart.

Cases in which the gangrene is due solely to heart debility should be recognised if possible, as the advisability of operating under these circumstances would appear to be very doubtful.

Another way in which the heart may be directly responsible is by embolism of an artery. The following is an example.

Case 12 suffered from mitral disease. Gangrene developed in the left leg and spread rapidly up to the thigh. After death there were found numerous emboli in the kidneys and spleen, and a clot in the lower part of the aorta which extended down both femoral arteries. Before death gangrene had commenced in the right foot.

In the majority of cases, however, of senile gangrene the obstruction to the blood-flow is mainly dependent on obstructed lumen of the large arteries of the leg, due either to atheroma, to obliterative endarteritis, to thrombosis, or allied conditions.

In thirty out of the forty-eight cases of the present series

some note is made pointing to diseased vessel-walls or obstructed lumen.

In thirteen cases it is noted that the lumen was obliterated (6, 10, 14, 16, 17, 18, 31, 32, 39, 40, 43, 44, 45). In Case 16 the permeability of the artery was destroyed by a thickening of its coats. In eight cases (6, 17, 18, 32, 39, 40, 43, 44) it is noted that the vessel was blocked by a clot. In the remainder it is stated simply that the artery was occluded.

In the other cases partial obstruction was brought about by thickening of the vessel-walls, or by the projection into them of rough calcareous plates tending to the deposit of fibrin.

As regards the site of the obstruction in cases in which the permeability of the vessel was noted as being destroyed, the following conditions were found either after amputation or death :—18, 39, 40, 43, femoral artery occluded by a clot, the site of the clot in Case 18 being the common femoral ; 6, clot in aorta and femorals ; 32, clot in the popliteal ; 14, 45, anterior and posterior tibial arteries occluded ; 16, 17, 31, posterior tibial occluded ; 10, anterior tibial occluded.

There are five cases in which it is stated that there did not appear to be any marked atheroma or thickening of the radial and temporal arteries (2, 19, 20, 25, 30). Of these, Case 2 had symptoms pointing to both diabetes and locomotor ataxy, and it is possible that in this case nerve degeneration may have been responsible.

That an apparently healthy radial or temporal artery is not to be taken as too certain a guide to the condition of the vessels of the lower limb is shown by four cases, where with good radial and temporal arteries there was obstruction in the arteries of the leg (14, 16, 18, 32).

Although arterio-sclerosis is the most important factor in the production of gangrene, in the majority of cases necrosis is actually initiated by the supervention of some local inflammatory disturbance. In consequence of the already hampered circulation, a very slight additional hindrance is sufficient to cause complete stasis. This inflammatory stasis may be the result of an injury, often exceedingly slight, such as walking an unaccustomed distance, a blow, or more often to some septic infection, either through a recent wound, such

as a cut corn, or nail, or through some pre-existent lesion, such as a perforating ulcer or ingrowing toe-nail.

In one case gangrene followed a supposed attack of gout (39).

In the above cases the symptoms are primarily those of inflammation. There is swelling with very often intense pain. The parts affected are first of all of a bluish-red colour, gradually becoming black.

Rarely there occurs what one might describe as a spontaneous origin. The patient notices a brownish or black spot on a toe, which gradually enlarges, leading to a mummification of the toe, without apparently any primary stage of inflammatory swelling and redness.

The commencement as bullæ on the feet in diabetics has been pointed out by Sir James Paget and others. Case 42 is an example.

Nerve degeneration.—The influence that nerve lesions alone are capable of exercising in the production of gangrene is difficult to estimate, as they are in most cases associated with other factors, such as arterial degeneration and toxic conditions such as occur in diabetes and Bright's disease.

In three cases the gangrene started in a perforating ulcer. Two of the cases (2, 26) were diabetic, one (3) was suffering from locomotor ataxy. In all three there was an area of anæsthesia on the affected foot, and in one case (2) the knee-jerks were slight. In none of the cases is there any note of the vessels, but we are scarcely justified in concluding that the arteries were healthy, as in two cases there was no operation performed, and in the third (26) gangrene recurred after amputation through the leg.

Indications for operating.—The points to decide are in what cases amputation is necessary, and if so when it should be performed.

With gangrene confined to the toes, with a line of demarcation forming, with no signs of extension of gangrene or septic inflammation to the foot, and with no symptoms of general poisoning, an expectant treatment may be adopted. Even when the toe separates spontaneously the results are, in many cases at all events, not particularly good, the wound repairing very slowly or a callous ulcer resulting.

In what proportion of cases of senile gangrene, mortification remains limited to the toes, and in what proportion successful separation of the necrotic tissues occurs, the data at command are scarcely sufficient to give anything like an accurate idea. The fact, however, remains that in the larger number of cases admitted into hospital, gangrene or septic inflammation spreads sooner or later to the foot.

The questions that arise when gangrene has reached the foot are these:

1. Is there a reasonable probability that a line of demarcation will form before the gangrene has extended very far, and that separation will take place before the powers of the patient are exhausted?

2. If, on the other hand, it seems more than probable that the patient will succumb before separation of the necrosed tissues takes place, when and where should amputation be performed?

3. Should the rule of waiting for a line of demarcation be followed?

The dangers that are incurred in the spread of gangrenous and inflammatory processes, or in the process of demarcation of the living from the dead tissues, should be borne in mind. They are shortly as follows:

1. Septic intoxication. The importance of this is shown by the frequency with which patients are affected with restlessness, delirium, and finally typhoid symptoms.

2. Septic infection of the neighbouring soft parts, leading to the formation of abscesses and the extension of the gangrenous process. I have already pointed how likely this is to occur when there is any tampering with the mortified parts, or where amputation close to the dead tissue is performed. There seems to be a very marked tendency under these circumstances to extension along the tendon sheaths.

3. Infection may be general, septicæmia and pyæmia resulting.

Gangrene having extended to the foot, we may consider under what circumstances and when amputation should be performed.

If, as sometimes happens, in the course of a slowly progressive gangrene or during the demarcation of the

dead from the living tissues, an acute phlegmonous or gangrenous process arises, there will be no question about operation. Amputation must be performed as rapidly as circumstances will permit.

A common course of events is as follows:—Gangrene having been for some time localised to the toes, spreads to the dorsum or sole of the foot, and extends slowly, being in places moist, in others dry, with considerable swelling of the foot, and perhaps of the lower part of the leg. Then when a line of demarcation is in course of formation, and hopes are entertained of a successful separation of the gangrenous parts, a fresh patch occurs, perhaps adjoining the sphacelus, or making its appearance on the heel. Or an abscess forms in the foot, and an incision made into this is followed by a fresh outburst of inflammation or gangrene. The patient is almost certainly by this time showing signs of poisoning. He is restless, delirious at nights, and worn out by pain. At this stage the patient comes under the surgeon's care. His general condition is passing from bad to worse, and no doubt can be entertained but that the sole chance of saving the patient's life is by prompt removal of the dead parts. Unfortunately the patient's vitality has by this time been so lowered that failure not infrequently results.

This leads us to a consideration of a third group, in which, gangrene having extended to the foot, the patient as yet shows no signs of poisoning. These are the cases that present the greatest difficulty. On the one hand, it might be urged that as the general symptoms are not pressing, an expectant policy might be adopted, and the gangrenous area encouraged to separate. On the other hand, if it can be shown that the chance of a successful separation taking place is small, there can remain no doubt but that the present is the time when an operation may be performed with the greatest probability of success. To answer this question of waiting it is sufficient to examine the records of the cases appended. In almost every case in which gangrene had spread to the foot, and in which the condition of the patient was not already beyond the powers of surgery to affect for the better, an operation was indicated, either

by the spread of gangrene or phlegmonous inflammation, or by symptoms pointing to poisoning of the system. What is specially noticeable is the small number of cases in which separation of any considerable portion of the foot had successfully taken place. Only two cases are recorded.

In an old woman of sixty-two (Case 9) gangrene made its appearance in the foot seven weeks before, as the result apparently of a chronic ulcer which had extended the whole way round the leg. The foot had separated above the ankle before admission, and from the granulating surface left protruded the necrosed ends of the tibia and fibula.

In the other case, that of a man of fifty-four (Case 25), the fore-part of the foot had been removed two years ago at another hospital. What are the results when separation has taken place? I have already pointed out how disappointing is the subsequent condition, even after separation of a toe, either very slow healing taking place or a chronic ulcer remaining. The results are not likely to be more encouraging when the mortified parts are larger in extent. Case 9, during the period of demarcation, had passed from a condition of delirium into one that was increasingly fatuous, and she left the hospital in the same condition as on admission, with a surface showing but slight power of healing, and exposed to all the risks of re-infection of the sound tissues. In Case 25 the surface of the stump had never healed over, and latterly the reappearance of a slough had necessitated an amputation, from which the patient rapidly recovered.

We may fairly conclude that the encouragement to wait is exceedingly small; that in most cases the patient dies of sapræmia or septicæmia, with, in all probability, an extension of the gangrene; that even if separation does occur he is left with a granulating surface of slow healing powers with its attendant risks.

The one conclusion left is that one is strongly justified in amputating the limb in all cases in which gangrene has spread to the foot, and the sooner this is performed the better the chance that the patient has of recovery.

As regards the site of amputation, enough has been said on that subject to point to the advisability of amputation

through or above the knee. The results of amputation close to the mortified parts are not such as would lead one to recommend that method of removal.

Kuster's experience is again worth quoting. Of thirteen cases in which amputation was performed below the knee, including Chopart's and Lisfranc's amputations, two healed, two died of gangrene of the flaps, and nine were re-amputated.

Of seventeen primary amputations through and above the knee, two healed by first intention, three after marginal flap gangrene, three after re-amputation, eight died.

Of ten secondary amputations through and above the knee, three healed by first intention, six after marginal gangrene, one after re-amputation.

In the present series, of ten cases in which amputation was performed below the knee, six died, two recovered, and two were re-amputated through the thigh.

Of eighteen cases of amputation through the thigh, there was primary union in four cases, in two others healing was good, four recovered after gangrene or suppuration in the wound, eight died.

In the case of embolism of an artery occurring in an old person in whom the arteries appear to be otherwise good, it might be considered justifiable to wait until the extent of the gangrene became known, to avoid amputating unnecessarily high in the limb.

It must be remembered, however, that such an occurrence in an old person,—and it is only such cases as would be included under the heading of senile gangrene that I am considering,—is likely to be complicated by thickened and atheromatous arteries, with imperfect readjustment of the collateral circulation. In these cases the indication for operation would be the same as those already considered for the forms of gangrene primarily dependent on arterio-sclerosis.

The advisability of amputating through the knee or higher up the thigh would be dependent on the condition of the circulation in the femoral artery.

Mr. Pearce Gould[1] having ascertained that pulsation was good in the arteries above the knee, successfully amputated through that joint.

[1] 'Clin. Soc. Trans.,' 1891.

Erichsen has laid it down as one of the golden rules of surgery that no operation should be performed till a line of demarcation has formed. Unless one waits for the line to form, Erichsen says, it will be impossible to say where the mortification will stop, and amputation will be performed either too high or too low. But the force of this law is lost if it be agreed that in case of an operation being demanded, an amputation through or above the knee should be performed.

Mr. Pearce Gould, in a case published in the 'Clin. Soc. Trans.' for 1891, did not hesitate to amputate for gangrene due to well-marked obliterative endarteritis, before demarcation had set in. He noted that the dead parts were a source of danger to the living, and that a certain degree of sapræmia was resulting from the continuity of the dead and living parts.

I have already pointed out how far success depends upon early operation, and the dangers to which the patient is exposed in waiting for the line of demarcation to form. The observance of this rule can only result in disaster in the majority of cases. Not much attention seems to have been paid to it in the successful cases, at all events, of the present series.

Contra-indications to operation.—Such obvious general conditions as are exemplified in Case 1, when as a consequence of fatty heart the circulation was so impeded that gangrene threatened not only in the other foot but in the hands and elbows.

Diabetic gangrene.—In the present series of cases I have included not only those forms of gangrene called senile, but those dependent on diabetes. Clinically there is the closest resemblance between the two diseases, the majority of cases of diabetic gangrene depending upon arterio-sclerosis. The importance of this as a cause of diabetic gangrene has been emphasised by Lancereaux, Israel, Heidenhain, and Fischer, amongst others. Heidenhain points out that diabetes is combined very frequently, and often at a comparatively early age, with severe arterio-sclerosis; and to this is due in most probability the majority of cases of gangrene of the lower extremities. In eleven cases with

diabetes he found atheroma seven times. Israel found atheroma in thirteen cases which he examined for this lesion out of twenty. Of the cases in this series seven out of ten showed arterio-sclerosis.

The similarity of the factors in the causation of both senile and diabetic gangrene points to the advisability of employing the same methods of treatment in the two allied conditions; and although the results are not very promising in diabetes, the cases that Herbert Durham and I have already collected show that they are not absolutely discouraging. There is the more reason for operating early in diabetic cases on account of their great liability to septic infection.

With imperfect histories there is great difficulty in saying which cases are primarily diabetic and which cases are glycosuric secondarily to the septic lesion.

The importance of noting this difference from the point of view of prognosis is shown by Cases 31 and 32, which both exhibited traces of sugar apparently secondary to the gangrene. In both, primary union occurred after amputation through the thigh. On the other hand, of four cases in which there is every reason to suppose that diabetes was present, and in which amputation through the thigh was performed, three died. The cases are shown in the following table.

Of the ten cases the notes of which lead one to think that they were diabetic, eight died. The cases are insufficient in number to draw conclusions from.

Causes of failure.—The causes of failure are obvious from what has already been said.

In the low operations failure results from impaired blood-supply, and re-infection of the wound from the proximity of the infected tissues.

In high operations, in spite of all precautions, the cases show that gangrene may recur in the flaps. A bloodless condition of the wound at the time of operation will point almost certainly to death of a portion of the flaps. In four cases (22, 27, 36, 42) it is specially noted that there was very little blood lost at the time of amputation. If the operation were being carried out low down, this absence

Cases with Diabetes.

No.	Sex and age.	Condition of vessels, &c.	Treatment.	Progress.	Result.
(2)	M., 72	Knee-jerk slight, &c.	Toe separated	...	Relieved.
(10)	F., 60	Arterio-sclerosis	No operation	Coma	Death.
(14)	M., 67	Diabetes 4 years; arterio-sclerosis	Phlegmonous inflammation spreading up leg	...	Death.
(17)	M., 68	Arterio-sclerosis	Dead parts pared with scissors	Spread of gangrene	Death.
(18)	F., 60	Diabetes 6 months; arterio-sclerosis	...	Spreading gangrene	Death.
(26)	M., 57	D. 15 years	Amputation through leg	Gangrene of flaps	Death.
(41)	M., 67	Well nourished	Amputation through thigh	...	Death.
(42)	M., 64	Emaciated; arterio-sclerosis	Amputation through thigh	Coma	Death.
(45)	M., 64	Diabetes 5 years; arterio-sclerosis	Pott's fracture; gangrene; Stokes-Gritti's amputation	Good union	Recovered.
(47)	M., 79	Arterio-sclerosis	Amputation through thigh	Shock	Death.
			Cases with Secondary Glycosuria.		
(13)	M., 64	...	Gangrenous toes removed	Extension of gangrene	Death.
(20)	F., 71	...	Amputation of toe	Want of repair	Relieved.
(31)	M., 64	Knee-jerk diminished	Amputation through thigh	Primary union	Recovered.
(32)	M., 72	Arterio-sclerosis	Amputation through thigh	Primary union	Recovered.

of bleeding would point to the advisability of amputating higher up on the earliest possible opportunity.

The statistics of Kuster showed that secondary amputations for gangrene are by no means as fatal as one might expect. Of the secondary amputations through and above the knee that he performed all recovered.

A frequent cause of failure and death is leaving the operation till the powers of the patient are exhausted by septic poisoning, or more rarely till general septic infection has taken place.

Patients seem to bear the amputation through the thigh well. Death from shock generally occurs only in those whose general condition was exceedingly bad at the time of operation, or in those suffering from diabetes. The chance of coma supervening in this latter case has always to be allowed for.

In spite of the bad condition of the vessels, secondary hæmorrhage seems to be of very rare occurrence. Mr. Hutchinson states that he was not troubled by this complication in any of his cases.

There was no secondary hæmorrhage in any of the cases of this series. In one it is stated that there was troublesome oozing following the operation.

Conclusions.

1. The most important factor in the production of both senile and diabetic gangrene is a diseased condition of the arteries of the lower limb.

2. With gangrene confined to the toes an expectant antiseptic treatment should be adopted.

3. The picking and cutting away of dead structures is specially to be avoided, on account of the liability to reinfection of the already ill-nourished tissues on the proximal side of the line of demarcation.

4. The same remark applies to amputation close to the affected area. Not only is there a strong chance that the wound will be infected, but the nutrition of the parts is, on account of the bad supply of blood, too impaired to allow rapid healing; a chronic ulcer is liable to result.

5. When gangrene has reached the dorsal or plantar surface of the foot a high amputation is indicated.

6. In amputating high up, not only theoretical considerations, but the practical results obtained by Kuster and Hutchinson, and those shown in this paper, lead definitely to the conclusion that amputation through or above the knee is the operation to be chosen.

Cases of Amputation

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
1	F., 67	1893, 168	Admitted Dec. 11th, 1893. Temperature between 98° and 99° throughout; radial arteries not atheromatous. Urine 1018; trace of albumen	Discoloration extending up to line of metatarso-phalangeal joints; toes cold; demarcation not marked; on inner side of 4th toe black leathery scab; dorsum of foot œdematous
2	M., 72	1890, 148	Admitted Feb. 22nd, 1890. Tenderness in calf muscles; knee-jerks present, but slight; cataract in right eye; pupils react to accommodation, but not to light. Pulse 84, regular, compressible. Urine acid, no albumen; sugar on admission 7·28 grs. per oz., diminishing under treatment to 3 grs. per oz.; it disappeared on March 3rd, but reappeared on the 12th	Left foot and leg swollen to a little above knee; ulcer on outer side of left 5th toe with irregular black edges and base covered by slough. Over proximal end of 1st phalanx a circular ulcer with raised edges of thickened white skin; anæsthesia along line of metatarso-phalangeal joints on dorsum of foot; partial anæsthesia along outer side of foot
3	M., 49	1890, 145	Admitted Aug. 25th. Syphilis 29 years ago; symptoms of locomotor ataxy	Small circular ulcer, deep sharp-cut edges, sloughy base, external to ball of left hallux; black patch on inner side of great toe; anæsthesia around gangrenous patch for 1½ inches
4	M., 83	1890, 149	Admitted Oct. 9th. 18 months ago had two paralytic seizures; demented. Urine 1015, acid, clear, no albumen, no sugar. Temp. 97°. Right-sided paralysis; can move leg a little, arm freely	Tips of toes and sole of left foot black and gangrenous; ulcer over front of ankle-joint, due to injury years ago, covered with dark green corneous matter; whole foot swollen and tender
5	F., 51	1888, 138	Admitted Sept. 19th, 1887. Very weak pulse; temp. 98·8°; pale, poorly nourished. Heart very feeble, irregular	Large gangrenous patch on dorsum and outer side of right foot, extending backwards over outer malleolus, 7½ inches long. Line of demarcation not yet formed. Swelling of foot and lower half of leg

in Senile Gangrene.

Mode of onset.	Progress and treatment.	Result.	Remarks.
Spontaneous origin 4 mos. ago in great toe	Dec. 18th.—Extension of gangrenous patch to 3rd toe; pulsation felt in anterior and posterior tibials; gangrene threatening in left foot. 26th.—Discoloration in hands and elbows	Death Dec. 26th	Gangrene due to fatty degeneration of heart; atheromatous aorta; granular kidneys; operation plainly contra-indicated on account of general failure of circulation.
Has for many years had corns on big and little toes; about 14 days ago great and little toes became painful and began to discharge, and leg became swollen and painful; knows of no injury	March 6th.—Circular ulcer on 1st toe scabbed over; sensation good everywhere except on 4th and 5th toes; line of demarcation formed over 2nd phalanx of 5th toe. 31st.—Slough separated; fresh patch of ulceration over 4th metatarsal bone. April 4th.—Ulcer healed; 5th toe granulating; 4th toe anæsthetic	Discharged Apr. 5th	Diabetes; corns; separation of little toe.
Corn over ball of left great toe, which he is in the habit of cutting; it cracked 4 years ago, and ulcer started, which became deeper and deeper. Two days ago he noticed black patch on inner side of great toe	Sept. 3rd.—Small ulcer on pinna of each ear. Hard black patch on head of 2nd metatarsal bone on plantar aspect (right side). Ulcer on left foot healing. 15th.—Right foot healed; ulcers on ears and left foot in process of healing	Convalescing	Locomotor ataxy; perforating ulcers; gangrene.
Foot run over 15 to 16 years ago, causing wound on instep which has never healed. The blackness of foot (his nephew says) has been coming on for years	Patient was demented, and his friends did not wish any operation, so he went out in much the same condition as on admission	Discharged Oct. 25th	No note of arteries, but paralytic strokes point to bad vessels.
Commenced a fortnight ago as small white spot on dorsum of foot, which gradually increased in size. Was under treatment 5 years ago for exophthalmic goitre	26th.—Pulsation in popliteal and posterior tibial can be felt; slough separating. Slough separated, and ulcer became covered by healthy granulations. Oct. 13th.—Skin-grafting. Wound was granulated over by Jan. 10th	Relieved	Old exophthalmic goitre; feeble irregular heart.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
6	F., 52	1887, 129	Admitted March 13th. Very stout woman. Feeble pulse, 132, very irregular; dilated capillaries on face. Urine 1022, alkaline, no sugar or albumen. Respiration rapid and shallow; temp. 100·8°	Left leg gangrenous up to its upper fourth; mainly dry, but a little moist on its outer side, on which side leg is lying; no smell. On outer side gangrene has reached level of external condyle. Lower half of thigh is brawny, swollen, and painful. Right foot and leg cold and bluish, but sensitive; pulse cannot be felt in right leg
7	M., 77	1887, 126	Urine: no albumen or sugar, 1023	Redness of foot; toes blue; no distinct line of demarcation; foot cold and very painful
8	M., 61	1886, 121	Feb. 9th, 1886. Very bronchitic; prematurely aged; very feeble; pulse weak	Right leg colder than left, and right foot œdematous; no loss of sensation in foot; great toe entirely gangrenous, showing tendency to spread on to dorsum but not on to plantar surface; little toe black nearly up to root, discharging on its outer side. On left foot 2nd, 3rd, and 4th toes are somewhat discoloured
9	F., 62	1885, 118	Feb. 4th.—No previous illnesses. Healthy-looking old woman	Stump of right leg shows tibia protruding, with articular surface and internal malleolus intact. External malleolus and 1½ inches of fibula have separated, extremity of fibula being rough and softened. Tibia bare for some distance, and presents a dry, black, extremely corrugated appearance; soft parts covered by fairly healthy granulations
10	F., 60	1885, 118	Aug. 22nd.—Has had rheumatism. Urine 1035, contains sugar. Anti-diabetic treatment. General health good. Amount of urine passed varied from 40 to 50 oz. in 24 hours. Sept. 3rd.—10·9 grs. per oz., trace of albumen, sp. gr. 1027. 8th.—7·39 grs. per oz., 1028. 10th.—12·1 grs. per oz., 1030, 40 oz.	Aug. 27th.—Red area around root of left 5th toe has not increased. Anæsthesia extends ½ inch beyond black slough. Foot no colder than other; fœtor slight

Mode of onset.	Progress and treatment.	Result.	Remarks.
Has had rheumatic fever twice; last time 4 years ago. Since then suffered with heart and bronchitis. Has been drinking heavily for last few years; raw spirits	March 17th.—Gangrene extending up to junction of lower and middle thirds of thigh. 22nd.—Spreading up inner side of thigh to upper third; mental condition impaired; temp. 100°; odour of leg very offensive. 25th.—Gangrene has now commenced in right foot	Death Mar.25th	P.M.—Clot in lower part of aorta extending down left femoral artery and for 2 inches down right femoral artery; mitral stenosis; recent endocarditis of mitral, aortic, tricuspid valves; emboli in kidneys and spleen; nutmeg liver.
—	—	Discharged *in statu quo ante*	—
12 weeks ago right great toe became swollen and inflamed; said by doctor to be a chilblain; toe very painful. 3 weeks ago toe began to turn black under the nail and discharge; nail came off, and whole toe turned black	Patient after admission was very restless; delirious at times; pulse about 100, temp. about 98°. He died on Feb. 15th. P.M.—Broncho-pneumonia and collapse; pus in tubes. Arteries very bad throughout body; calcareous coronary arteries; femorals lined with calcareous deposit; heart-muscle bad; right ventricle hypertrophied. Gangrene of 1st and 3rd right toes spread on to dorsum; gangrene spread up left leg on inner side; gouty deposit in interphalangeal joints	Death	General condition too severe to admit of operation; severe general arterio-sclerosis.
Following a blow 6 years ago an ulcer developed which spread half-way up leg and all round it. About 7 weeks ago noticed foot was getting dusky, and a week or two later it rather suddenly turned black. 3 days ago foot dropped off	9th.—Delirious; temp. 101·8°. 17th.—Pulse 132, soft and weak; condition increasingly fatuous and imbecile; no extension of gangrene; part looks weakly; bone absolutely fixed. 19th.—Urine 1020, alkaline, no albumen, no sugar. 21st.—Discharged in above condition	Discharged *in statu quo ante*	Spontaneous separation of foot.
5 weeks ago what seemed to be a soft corn appeared on 5th toe. It opened and became painful, nail soon came off, and it began to turn black	Sept. 8th.—Distal phalanx nearly separated. 10th.—Breathing noticed in night to be rapid. 12th.—Respiration became laboured; patient became restless and delirious; appears dull, but answers questions rationally. Resp. 32, deep and rather noisy. 11 p.m.—Pulse 112, feeble and irregular, intermittent; surface of body cold; pulse in radial and posterior tibial arteries can be felt; no bruit heard; chest resonant; air enters bases freely	Death	Diabetes; coma. P.M.—All vessels calcareous. Anterior tibial artery on left side occluded; posterior tibial pervious; kidneys slightly enlarged.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
11	M., 44	1881, 93	Jan. 22nd. — Heart condition seems to point to adherent pericardium; no rheumatism; tanner	Aching pain in left ankle and inner side of leg; cannot walk far without being seized with cramping pain in calf. No pulsation in anterior or posterior tibials; dorsum of foot presents purplish discoloration; toes cold; considerable loss of sensation in 1st, 3rd, and 4th toes
12	F., 76	1881, 93	June 8th.—Tongue hard and dry; heart feeble and intermittent; bruit heard. Temp. 97°, pulse 104. Radial artery hard and cord-like, large and tortuous. Urine all passed under him	Gangrene of left foot extending up to malleoli, with a dusky mottled area extending up another 3½ inches. Right foot almost entirely gangrenous; moist in parts on dorsum
13	M., 64	1879, 80	July 3rd, 1879.—Urine 1010, contains small amount of sugar. Aug. 1st. — Small amount of sugar; mitral systolic bruit	Anterior half of left foot gangrenous, with line of demarcation; foot red and swollen beyond line of demarcation. Posterior tibial pulsates very feebly behind internal malleolus
14	M., 67	1893, 167	June 1st, 1893.—Glycosuria 4 years ago when in hospital for fistula in ano. Febrile condition; temp. 100·2°; pulse soft and compressible. Polyuria, sugar 3 grs. to oz., 1020, acid	Terminal phalanx of 3rd toe sloughed off; rest of toe moist, black, and foul. On sole just below gangrenous toe is a reddened area; lower two thirds of leg and foot œdematous

Mode of onset.	Progress and treatment.	Result.	Remarks.
Was seized 14 months ago on getting out of bed with severe shooting pains, extending from ankle up outer side of leg and inner side of thigh to groin.	By Feb. 8th pulsation had returned in arteries and sensation in the toes, and he went out well Feb. 12th. Remained in bed 8 weeks, and did not leave house for another 11 weeks. Unable to do any work for 7 months, and then only light work. At Christmas the old trouble returned. At Out-patient's on Jan. 15th 2 outer toes were dusky and exceedingly cold; no pulsation in posterior tibial; dorsum of foot œdematous and discoloured	Recovery	Threatening gangrene with loss of pulsation in tibials. Return of pulsation.
8 weeks ago taken ill with palpitations. Shortly after this had shooting pains in feet. Blisters and poultices applied; feet began to swell, and about a fortnight later turned black	June 12th.—Condition rapidly becoming worse; patient in a typhoid state. Pulse 128, irregular. P.M.—Mitral valve thickened; calcareous plates in thickened arterial walls	Death June16th	Mitral disease; gangrene both feet; arterial sclerosis.
Corn on outer side of left little toe made to bleed by man treading on it; toe inflamed; foot gradually became gangrenous	July 4th.—*Three outer toes removed* without pain. 9th.—Local condition improving; line of demarcation more marked; granulations appearing. 21st.—Great toe and half of 4th metatarsal bone removed with some amount of slough. 28th.—Gangrenous patch appeared on heel; anterior part of foot doing well. Aug. 4th.—Anterior part of shafts of 2nd and 3rd metatarsal bones removed. Aug. 12th.—Anterior part of 1st metatarsal bone came away. Oct. 1st.—*Sloughing tendon cut away;* subsequent extension of inflammation along course of tendon; pus let out by laying open sheath. 23rd.—Erysipelas extending up leg. 27th.—Patient died with signs of pneumonia	Death	Glycosuria; amputation advised on July 28th. P.M.—Femoral artery thickened and calcareous; pneumonia at left base.
Spontaneous a fortnight ago; no abrasion or traumatism known; toe became black and foul in a few days	June 3rd.—*Abscess in sole incised.* 8th.—Abscess on inner side of tendo-Achillis opened; foul pus and gas escaped; abscess communicated by track with abscess in sole. 10th.—Infective process rapidly spreading up leg; there is gas almost as high as knee; pocket of pus on outer side of leg incised; general condition fairly good. 11th.—Suppuration extending; amputation advised. 13th.—Patient drowsy, restless; pulse 120. 14th.—Semi-comatose; pulse 150. Death	Death	Diabetes; phlegmonous inflammation; staphylococci in pus. P.M.—Kidneys large and pale; anterior and posterior tibials obstructed.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
15	M., 71	1892, 161	July 22nd, 1892.—Stout florid man; temp. 100°; urine clear, no note of albumen or sugar in it	Left great toe black in colour; fluctuating swelling in ball of great toe; shooting pain
16	M., 73	1883, 105	Nov. 14th, 1883.—Pulse 100, resp. 36, temp. 103·5°. Has lived a temperate life. Urine passed under him	3 inner toes of right foot gangrenous; some blebs on dorsum just above heads of metatarsal bones; on removal of these, subjacent parts are seen to be reddish green. Bleb just below external malleolus. Inflamed area extended up to ankle-joint
17	M., 68	1883, 104	Feb. 21st, 1883.—Fairly healthy-looking man, though apparently a drinker. Tongue dry, furred; pulse regular; arteries hard and unyielding, like rigid tubes. Respiration and temperature normal. No previous history of diabetes. Sugar noted in urine on March 2nd and March 5th	Gangrene of left hallux and 2nd toe; black, cold, dry, insensitive, and very foul, with thin, blackish, foul discharge; distinct line of demarcation (ulceration); inner side of foot swollen and discoloured
18	F., 60	1881, 93	June 22nd, 1881.—Has taken more to drink and passed more urine than usual since Christmas; patient is very weak, chilly, breathes heavily; tongue dry and rough; pulse weak and small, but artery does not appear thickened. 24th.—Urine: large amount of sugar; sp. gr. 1042. 27th.—80 oz. urine in 24 hours; contained 4375 grs. sugar; anti-diabetic treatment. Carboluria on July 14th	Right 4th toe gangrenous; front half of dorsum of foot swollen; an *incision* made into this; a quantity of foul pus escaped

Mode of onset.	Progress and treatment.	Result.	Remarks.
Some months ago had ingrowing toe-nail; drew blood in cutting it; suppuration took place; a fortnight ago a piece of dead bone was removed from toe	22nd.—*Abscess opened;* thin watery pus escaped. 26th.—Vomiting; difficulty in passing water. 27th.—Gangrene spreading fast	Death July 29th	—
3 inner toes grazed by railway van	Nov. 20th.—3 *inner toes removed,* with slough over 3 inner metatarsal bones. This surface is granulating; area of swelling has extended up over lower third of fibula; temp. keeps high, 100°—103°; pulse satisfactory, 70—80, fairly strong. 22nd.—Restless and delirious. 23rd.—Ulceration on dorsum has extended up further towards ankle; tendons on dorsum partially exposed. No extension as yet to sole. Restlessness and delirium continued. Temp. 99°—101°, pulse 70—80, resp. 30—35. 28th.—Has slept continuously since 4 o'clock yesterday. Temp. 103·2°, resp. 55, pulse 108. 29th.—Death	Death	Amputation not deemed advisable on account of patient's restless muttering delirium, tendency to knock his limbs about, and the passing of his excreta under him. P.M.—Right femoral calcareous; posterior tibial in one place occluded by thickening of wall; dorsalis pedis occluded; aorta and radial healthy; calcareous patch on mitral valve.
Has had a bunion for some years on inner side of metatarso-phalangeal joint of left great toe. Became inflamed last Christmas. It burst after 7 days, discharging pus. He aggravated this by walking about, and 3½ weeks ago instep of left foot became much inflamed, and he noticed a numbness of sensation; great and 2nd toes rapidly became discoloured	Feb. 21st.—*Dead skin at cleft between 1st and 2nd toes pared away* by scissors and forceps. March 3rd.—Fresh speck of gangrene on back of great toe. 5th.—Hæmaturia in large amount; gangrene spreading. 8th.—Has been gradually sinking. Death	Death	Alcoholic; ? diabetic. P.M.—Atheroma of anterior and posterior tibials; plug found at bifurcation of plantar arteries; pneumonia at lower lobe of left lung; patches of sloughing in mucous membrane of bladder, and early peritonitis. Kidneys 15 oz.
About 6 months ago felt pain on under surface of 4th toe; it used to bleed. Last Whitsuntide she thinks she poisoned toe with a pair of new stockings; from that time it became worse, and leg began to swell	July 7th.—Incision on dorsum of foot healed; 4th toe nearly separated; 3rd toe gangrenous. 20th.—Two gangrenous patches on outer side of foot; 4th toe separated; line of demarcation round base of 3rd toe. Gangrene spread gradually; by Aug. 20th the remaining toes were discoloured; by 31st there were several black patches on foot; foot and leg cold. Sept. 2nd.—Black patches round knee and lower part of thigh. Death Sept. 5th	Death	Diabetes. P.M.—Atheroma of aorta; clot at point of division of common femoral. Kidneys 11 oz., full of emboli. Ulcerative colitis and peritonitis.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
19	M., 75	1880, 86	June 5th.—Radial artery not hard	Terminal phalanx of 4th toe gangrenous; leg and foot swollen
20	F., 71	1890, 149	March 3rd.—Fairly well nourished; does not eat much, but says she is always thirsty. Radial and temporal arteries do not appear to be atheromatous. Urine contains sugar, no albumen. Sugar not found on April 4th. No rise of temperature. Pil. Opii	Right hallux black and shrivelled; line of demarcation over centre of 1st phalanx; edge of living tissues red and raised, and showing points of slough; redness along inner border of foot to centre; sensation elsewhere than in toe good
21	M., 62	1883, 103	Mar. 20th.—Pale and weak looking. Temp. 102·2°, pulse 96; artery hard and cord-like; resp. 18; urine, no albumen or sugar	Right foot swollen and hot; nearly whole of sole undermined; great toe abducted, with slough over its metatarso-phalangeal joint; slough on dorsum of foot. Several sinuses on under surface of great toe and sole of foot; bare bone to be felt; foul discharge
22	M., 60	1880, 86	May 14th.—Thin weak man, publican; says he is temperate. Radial artery hard and tortuous; calcareous plates felt; no albumen or sugar in urine	Gangrenous patch spreading up dorsum of left foot, and exposing tarso-metatarsal joints; pain in foot

Mode of onset.	Progress and treatment.	Result.	Remarks.
Noticed a swelling of 4th toe 4 months ago, which broke and festered	June 9th.—*End of 4th toe snipped off.* 14th.—Head of 2nd phalanx removed with bone forceps. July 6th.—Abscess has formed in sole. 17th.—Gangrene spreading all over foot, exposing cuboid and flexor tendons. 26th.—Death	Death	P.M.—Broncho-pneumonia.
2 years ago after walking felt pain in toe, and noticed it to be red and shiny; toe has since been getting gradually worse; last Christmas it cracked	9th.—*Great toe amputated* at metatarso-phalangeal joint. Wound showed subsequently but little power of repair; sloughing of edges. A black spot was noticed on each heel on March 22nd; these did not spread much. Apr. 4th.—Heels have nearly healed up; 2nd left toe becoming discoloured. 30th.—Toe quite black at end. May 26th.—Patient discharged in above condition, amputation wound showing but little repair	Discharged	Glycosuria; want of repair after amputation of toe.
For last 15 years left leg has been in habit of swelling if he walked much on it. Jan. 10th.—Walked about all day looking for work; afterwards felt great pain in right great toe; went to a doctor, who said it was a corn, and cut it with a knife; a week later an opening appeared at site of corn, which discharged	Apr. 2nd.—*Amputation of great toe* through metatarsal bone. Same evening temp. 105°, pulse 90. 7th.—Patient been low and delirious for last day or two; patch of gangrene on external malleolus, 2 inches in diameter. Temp. 97°—103°. 12th.—Very delirious; gangrene extending along outer side of leg in course of peronei muscles, which are sloughy. 17th.—Gangrene has extended almost to knee. Patient died: aorta thickened; radials and other large vessels thickened. Kidneys 8½ oz.	Death	Amputation of toe; spreading gangrene.
2 years ago after walking 9 or 10 miles he found 3 outer toes of left foot swollen, and on inner side of middle toe a black spot, which became a small discharging hole. *Middle toe was amputated* by a doctor; 3 days later gangrene set in, and he came to hospital. Soon after coming in his 2 outer toes dropped off. He went out with wound unhealed, and at the end of 8 months the wound began to slough	June 8th.—*Syme's amputation;* no spurting from arteries on removal of pressure from femoral; they gaped, and a small quantity of blood trickled from the wound. June 12th.—Flaps sloughing; gangrene spread 2½ inches in front, 5½ inches behind. Died June 14th	Death	Recurrence of gangrene after amputation of toe; Syme's amputation; recurrence of gangrene; pyæmia.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
23	M., 58	1879,	Sept. 5th, 1878. — Considerable atheroma of radial arteries	Large sloughy surface extending as far back as metatarso-phalangeal joints of 2nd, 3rd, and 4th toes (left); flexor tendons exposed
24	M., 45	1879, 81	Feb. 5th.—Syphilis several years ago. Has been a fireman, and exposed to heat and cold. Urine 1020, no sugar, no albumen; tongue red and moist; liver and spleen enlarged; liver dulness 2¼ inches below margin of ribs; spleen nearly to umbilicus; no heart murmur; heart action quiet	Great toe black, loose at metatarso-phalangeal joint; mottling of 2nd toe; no line of demarcation
25	M., 54	1892, 161	Admitted March 14th. General health good; temporal and radial arteries not particularly atheromatous; knee-jerk present and normal; heart-sounds normal; no enlargement of heart. Urine tested many times; no albumen, sugar, or blood; acid; 1022. Temp. about 99°, never much above	A yellow slough covering stump at site of removal of fore part of foot
26	M., 57	1891, 155	Admitted July 22nd, 1891. Diabetic history of 15 years; polyuria; sugar; cataract. Cramp, coldness, and numbness in legs for last 5 months, with pain in feet, especially in left foot; ulcer on left heel soon after. On admission lower limbs wasted and cold	Perforating ulcer on right foot between 4th and 5th toes; is deep, with black slough covering its base; surrounded by wide inflammatory area, which extends on to dorsum of foot. Sensation impaired on plantar surface of right foot. Oct. 8th.—Gangrenous patch on plantar surface of great toe; no sugar now in urine; subsequent extension of gangrene to sole of foot
27	M., 76	1888, 138	Aug. 10th, 1888. — Pigmented area round lower part of left leg; no history of ulceration; foot warm; no pulsation felt in tibials; temp. 99°—104°. Heart-sounds good, but radial artery badly filled	Whole of left foot swollen, red, brawny, and œdematous as far as ankle; yellow patch of slough extending along outer side of foot from base of 3rd and 4th toes to within short distance of ankle; pain much less than it was

Mode of onset.	Progress and treatment.	Result.	Remarks.
4 years ago noticed 3rd left toe was red and inflamed; it became worse, suppurated, and was amputated	Oct. 7th.—*3rd toe removed* just above head of metatarsal bone, and 4th toe through 1st phalanx; a little bleeding, but not much pain. Nov. 7th.—Wound still discharging; proximal end of 1st phalanx came away. Dec. 18th.—Small patch of sloughing on under surface of foot. Mar. 14th.—Ulcers have healed over with exception of very small spot	Recovered	Amputation of toes; very slow healing (5 months)
2 years ago plate of iron fell on his foot; very little pain at time, but soon after the little toe became gangrenous, and was *amputated*. 7 weeks ago noticed his foot was cold, and soon afterwards the great toe turned black	Mar. 8th.—*Lisfranc's amputation.* Wound had healed by May 27th	Recovered	Syphilis; Lisfranc's amputation; prolonged healing.
In Oct., 1889, a soft corn between the 4th and 5th toes of right foot was treated with acid. From this time the foot began to be cold, and to blacken on its outer side. Finally foot became black as far as ankle, where line of demarcation formed; foot removed without anæsthetic (May, 1890) at Charing Cross Hospital. Foot never entirely healed, and has become worse during last 2 months	March 20th.—*Amputation at seat of election.* Examination of limb showed superficial ulceration involving only skin and subcutaneous tissue; bones healthy; no note of arteries. Wound healed well, and he went out April 15th with stump quite healed	Recovery	Removal of fore part of foot for gangrene; persistent ulceration and sloughing; amputation through tibia; good union.
4 weeks before admission noticed an ulcer between 4th and 5th toes on right foot, which was excessively painful	Oct. 30th.—*Amputation at seat of election.* Wound first looked as if it was healing by primary union, but it subsequently broke down, and gangrene appeared on the outer flap. This extended, and death occurred on Nov. 8th. P.M.—Atheroma of aorta and pulmonary arteries; granular kidneys	Death	Diabetes; perforating ulcers; gangrene; amputation at seat of election; recurrence of gangrene in flaps.
16 years ago two toes (1st and 3rd right) were removed by Mr. Birkett for gangrene. 3 weeks ago toes of left foot became swollen and red, with much pain; redness has gradually spread	13th.—Tongue dry, brownish red, cracked; marked spread of gangrene; area of sloughing and burrowing suppuration over outer two thirds of dorsum of foot. 14th.—*Amputation by lateral flaps in upper third of leg; remarkably little bleeding.* 15th.—Did not suffer much from shock of operation; tongue moister and cleaner; stump dressed on account of free sanguineous oozing; no signs of gangrene; flaps look well. 16th.—No union between flaps. 17th.—Flaps gangrenous; general condition much worse. 19th.—Patient died. P.M.—Pyæmia; cirrhosis of liver	Death	Amputation through upper third of leg; gangrene of flaps; alcoholic.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
28	M. 54	1887, 129	Admitted August 24th, 1887. Urine acid; no albumen or sugar	Gangrene of 3 inner toes of left foot, parts of 2nd and 3rd being lost. Tendon of extensor longus hallucis exposed; line of demarcation opposite heads of metatarsal bones of 2nd and 3rd toes
29	M. 71	1882, 99	Admitted Nov. 29th, 1881. Arteries atheromatous; patient passes his excreta under him. Dec. 2nd.—Tongue large, red, and clean. Pulse 120, feeble, intermittent; both radials are calcareous	Foot gangrenous; line of demarcation extending from heel over front of ankle and lower third of fibula; foot black and shrivelled
30	M. 66	1880, 84	Admitted Jan. 12th. Arteries seem fairly healthy; urine healthy	Right leg and foot œdematous; great toe gangrenous; line of demarcation marked at base; black patch on dorsum of 1st phalanx of 2nd toe; gangrenous patch on dorsum of foot

Mode of onset.	Progress and treatment.	Result.	Remarks.
Commenced Nov., 1886, as a swelling on dorsum of foot which spread down to toes, following a twist of the foot. In Dec., 1886, a portion of 2nd toe came away	Sept. 15th.—Black circular patch on heel. 16th.—Abscess opened beneath patch on heel. 21st.—Another patch on outer side metatarso-phalangeal joint of 5th toe. 27th.—Part of 2nd phalanx of 2nd toe removed. Oct. 13th.—Great toe removed. Oct. 17th.—Hole in heel seems deeper, and looking less well. 22nd.—Heel granulating up; suppuration about parts from which toes have come away; heads of metatarsal bones exposed; tendons sloughing. Nov. 1st.—Suppuration extending along tendon sheaths to sole of foot. 4th.—Dark patch on ball of foot, from opening in which grumous pus is discharged. Temp. 100·2°; pulse 120. 15th.—*Amputation through leg* by antero-internal and postero-external flaps. Bones sawn through 3 inches below tubercle of tibia. 18th.—Some sloughing about edges of flaps. Pulse stronger, 100; temp. 98°—100°. 30th.—No more slough; flaps retracted; bone projecting and white. Since the 21st he has been suffering from symptoms pointing to lung mischief at left base. Dec. 7th.—Chest aspirated; 2 oz. of pus with foul air withdrawn. 8th.—Incision made over 9th space, and cavity in lung about 6 inches in diameter opened up, from which foul pus came. 9th.—Patient died	Death	Amputation through middle of leg; sloughing and retraction of flaps; necrosis of end of tibia; pulmonary infarct and gangrene of lung.
He was picking currants on a very hot day, when he felt a pain in his head. That night he lost the use of his leg. When he recovered from this attack he noticed a swelling like a soft corn between 2 toes; this broke, and the foot became purple and gangrene spread from the great toe up foot	Dec. 2nd.—Both malleoli exposed in area of demarcation. 5th.—Dead tissues separated through transverse tarsal joint, ligaments and tendons being cut with scissors; astragalus pulled out with fingers; further portions of slough removed. Jan. 6th.—Ulceration extending up leg; *amputation through upper third* of leg by lateral flaps; popliteal artery calcareous. 11th.—Stump gangrenous; foul. Patient gradually sank, and died Jan. 16th	Death	Paralysis; separation of dead tissues at transverse tarsal joint; extension of gangrene; amputation through leg; gangrene of stump; vessels atheromatous.
5 months ago inflammation by side of nail of right great toe. He got some dye into it, and observed a blackness extending backwards, involving gradually the whole toe. Intense pain; worked for 7 weeks; has been laid up for 11 weeks	The gangrene on dorsum, of moist variety, spread gradually till it met that of the toes. By Jan. 26th it was spreading on sole; 3 inner toes dry and black. Feb. 9th.—Metatarsal bones exposed on dorsum; in the sole gangrene is extending deeply. 16th.—Emphysema on outer side of heel and ankle; incision made into it.	Death	Spreading gangrene; incision in foot; rapid spread; amputation lower third of leg; gangrene of flaps.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
31	M. 64	1895	Admitted Dec. 28th, 1894. Temp. 100°; pulse 100. Heart-sounds good; knee-jerks both present, but not so well marked on affected leg as on sound one; pupils contracted, react neither to light nor accommodation. Urine acid, 1030, small amount of pus. Jan. 3rd.—Small amount of sugar. Patient has never suffered from thirst or polyuria	All the toes of left foot gangrenous down to the roots but the 2nd; great toe is quite dry; much pain
32	M. 72	1895	Admitted Nov. 3rd, 1894. Thin and ill-nourished. Habitually drinks 2 to 2½ pints of beer daily, with a little whisky. Suffers from enlargement of the prostate, with recurring attacks of hæmaturia for the last 7 years. Urine acid, 1018; contains pus, blood, and albumen; a little sugar on admission, none subsequently. Heart-sounds normal; arteries soft, and pulse is fair	Foot gangrenous as far as ankle; toes mummified; gangrene moist towards ankle; well-marked line of demarcation across ankle-joint in front, extending down in front of heel; complete anæsthesia of foot; fœtor; excruciating pain

Mode of onset.	Progress and treatment.	Result.	Remarks.
Scratched top of toe about 2 months ago. It afterwards became swollen and inflamed. About 10 days ago, whilst lying up with influenza, he found toe turning black	Jan. 3rd.—Temp. 99·5°—101° since admission. Gangrene has spread to 2nd toe and up on to dorsum, affecting outer two thirds of lower half of dorsum. 4th.—*Amputation* by circular incision carried round just above patella. Bone sawn through at about 4 inches from its lower end. Drainage-tube removed next day. Wound healed by primary union, with the exception of point of entry of drainage-tube; a small slough was removed from this corner of the flaps on Jan. 20th. Patient went out well on Feb. 21st, having gained flesh. On examination of leg after removal it was seen that, though there was a well-marked line of demarcation across the centre of foot, the tissues above this as far as ankle were bluish-looking, and were infiltrated with inflammatory exudation. No gangrene in sole of foot; arteries rigid and calcareous. Lumen of posterior tibial obliterated an inch and a half below bifurcation of popliteal. This was brought about partly by extensive thickening of the walls, partly by a firm decolourised ante-mortem thrombus. Vessel in its whole extent matted to surrounding parts; muscles on back of leg soft and devoid of colour. Those on front are much better nourished, and anterior tibial artery is patent	Recovered	Glycosuria; amputation through thigh; primary union with exception of very small slough; extreme atheroma of arteries.
Fell out of bed on Oct. 1st in a fit, and bruised the dorsum of foot. Foot became excruciatingly painful, and began to blacken from the toes upwards	Nov. 5th.—Delirious and restless. Urine porter-coloured. 16th.—Blood in urine has now disappeared; line of demarcation is deepening; general condition is better than on admission, and is thought to be good enough to warrant operation. *Amputation by circular incision just above patella,* with short vertical incision on outer side of limb. Bone sawn at junction of middle and lower thirds. 20th.—Stump dressed for first time; primary union had taken place. Dec. 16th.—Urine: sp. gr. 1006; more albumen than can be accounted for by pus. 23rd.—Right foot cold and painful; anterior tibial pulse perceptible, but weak. Discharged Jan. 3rd. Arteries of limbs removed were very good; nowhere calcareous. Firm ante-mortem clot in popliteal artery, firmly adherent to the walls, but apparently not quite occluding the lumen; no thrombosis elsewhere	Recovery	Bright's disease; enlarged prostate; amputation through thigh; primary union; popliteal artery thrombosed.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
33	M. 57	1894	Admitted Oct. 4th, 1894. Plantar reflexes and knee-jerks present equally on both sides; arteries thick and extremely hard; no pulsation felt in arteries below knee; no bruit. Temp. 99°—101°. Urine 1020; no albumen or sugar. 11th.—Trace of albumen. He appears to have drunk considerable amounts of alcohol	Right foot: 2nd toe black, a little moist at its base; 3rd toe black at tip, red elsewhere; other toes bluish; triangular black patch on dorsum of foot, extending from bases of toes upwards for 3½ inches; gangrene spreading into sole of foot
34	M. 55	1894	Dec. 9th, 1893.—Thin and emaciated; temp. 98°; pulse good; does not feel very ill	Gangrenous area on leg, extending on outer and back part of leg from a point 3 inches below knee to within 2 inches of ankle; line of demarcation; very foul smell
35	M. 69	1893, 166	Nov. 21st, 1892.—No note of general condition	Traumatic separation of 1st phalanx of great toe; toe amputated at metatarso-phalangeal joint
36	M. 60	1892, 162	Admitted Sept. 1st. Till 10 years ago was in the habit of drinking 3 to 4 quarts of beer daily; abstemious for last 10 years. For last few years has been working with feet under water (dock labourer). Urine 1020; no albumen; no sugar	Gangrene of left little toe, dry; well-marked line of demarcation; outer half of anterior half of dorsum of foot red, swollen, and œdematous. No pulsation felt in popliteal or tibials, very slight in femoral artery; in right leg pulsation could just be felt in tibial arteries

Mode of onset.	Progress and treatment.	Result.	Remarks.
Has had very cold feet for several years. Between Christmas and Easter began to suffer from pain in right calf and numbness in right leg. Soon after he became tottering in his gait. He made a corn at the end of his 2nd toe bleed 4 weeks ago; a week later he felt pain, and found his toe had turned black	Oct. 5th.—Patient delirious. 12th.—Restless and sleepless; temp. varying between 99° and 100°; pulse 80—90. Gangrene has reached ankle; patch on heel; swelling of leg. *Amputation through thigh* an inch above condyles; popliteal artery very rigid. Patient was restless and a little delirious for a few days after operation. With the exception of about 1½ inches the wound healed by primary union. He was discharged well on Nov. 16th. Arteries in limb removed were rigid tubes throughout; extremely calcareous; lumen of popliteal obstructed by projection of calcareous masses, some the size of a split pea, into the interior	Recovery	Alcoholic; ? neuritis; amputation an inch above condyles; obstruction in popliteal artery.
Knocked his leg against a poker 3 weeks ago; only a slight wound, skin-deep, was made. He walked about on it in spite of the pain he felt. Inflammation set in and gradually spread, followed by discoloration of the affected parts	Dec. 9th.—*Amputation through thigh*; bone sawn at junction of middle and lower thirds; femoral artery very atheromatous. Wound was plugged with gauze. 11th.—Gauze removed and stitches inserted. 19th.—Wound dressed. Healed by primary union	Recovery	Gangrene of leg; amputation through thigh; primary union.
Toe caught between footboard and platform in leaving train, and terminal phalanx of great toe amputated	Nov. 28th.—Skin of flaps black and gangrenous; patch of gangrene on outer side of foot. *Amputation through lower third of femur.* Wound did not do well; suppuration and sloughing. Dec. 15th.—Small black patch on inner side of flaps; this separated. Healing was prolonged. By June 14th flaps had firmly united, and patient went out well	Recovery	Amputation of toe for injury, gangrene occurring; amputation through thigh; recurrence of gangrene; slow healing.
No history of injury. 3 weeks ago noticed 2 last phalanges of little toe discoloured. Toe gradually became black	Sept. 6th.—*Amputation through transverse tarsal joint.* On removing the tourniquet it was found that very little blood was escaping from wound. Later in the day *re-amputation* was performed, and as no pulsation could then be felt in femoral artery leg was removed by sawing through femur at base of great trochanter. P.M.—Abdominal aorta and iliac arteries atheromatous; lungs contained much broken-down blood; left femoral artery thrombosed; gangrenous condition of stump, and bullæ on buttock; kidneys and heart normal	Death, Sept. 9th.	Amputation through transverse tarsal joint; re-amputation through upper part of thigh; gangrene of flaps; femoral artery thrombosed.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
37	F. 58	1892, 161	Admitted Oct. 26th, 1892. Had a carbuncle on back some years ago. 3 years ago a box of oranges fell on foot. She has never had the same use in it since. Urine 1022, acid, trace of albumen, no sugar	3 red circular scars on inner side of left foot, the result of previous ulceration. Posterior half of sole of left foot has sloughed away, exposing tarsal bones, extending on either side to within an inch of malleoli; good line of demarcation; brownish foul discharge. Superficial patch of gangrene on left heel
38	M., 54	1892, 161	Admitted June 15th, 1892. Urine 1010, alkaline, no sugar, trace of albumen; pus due to cystitis. Bladder washed out; general condition very bad on July 18th; temp. 101°; pulse hardly felt. Subsequent improvement	Gangrene had involved the whole of dorsum of foot; ulcer on lower part of leg and inner surface of foot, showing no gangrenous appearance; foul purulent discharge
39	M., 65	1890, 149	Feb. 26th.—Well nourished and of healthy appearance; urine 1028, acid, no albumen or sugar	Left 2nd toe black down to its base; foul smell; much pain; no loss of sensation except in gangrenous portion; small gangrenous patch on heel. No pulsation felt in arteries below the common femoral
40	M., 73	1889, 140	Sept. 18th, 1888.—Feeble-looking old man, complaining of sharp shooting pain in right calf and pricking sensation in left foot; great pain, preventing sleep. Urine 1016, no sugar, no albumen	Right great toe entirely gangrenous, the end being dry and shrivelled, the rest moist and purple. No pulsation in right posterior tibial artery; feeble pulsation in left posterior tibial

Mode of onset.	Progress and treatment.	Result.	Remarks.
Middle of last March noticed a black spot on inner side of left ankle, which has been increasing in size ever since. This, she thinks, originated in a chilblain "into which the frost got"	Oct. 28th.—*Stokes-Gritti amputation.* Nov. 14th.—A quantity of brownish discharge. 22nd.—Wound healing but slowly; transferred to infirmary	Recovery	Stokes-Gritti amputation; suppuration in wound; slow healing.
12 months ago noticed pimple on left instep; this broke, giving rise to an ulcer the size of a shilling, which was very painful. A few days later he noticed great toe was becoming black and dry	June 24th.—*Amputation through lower third of femur* by long anterior and short posterior flaps. July 1st.—Gangrenous patch in anterior flap cut away; wound sloughy; considerable amount of discharge. 4th.—More gangrene of anterior flap; end of bone protruding between flaps. 10th.—Almost entire absence of repair; flaps gape widely; end of femur dry and greyish-white for 1½ inches; few feeble granulations on posterior flap, none on anterior one. 15th.—Posterior flap covered with granulations; middle part of anterior flap cold and gangrenous. 16th.—Gangrenous portion removed. 18th.—Piece of femur 1½ inches long removed. 31st.—General condition improving; flaps covered with healthy granulations. Aug. 15th.—Wound continues to heal up steadily though slowly; discharge	Recovery	Amputation through femur; arteries extremely calcareous at site of amputation; recurrence of gangrene in flaps; very slow repair.
11 weeks ago had an attack of "gout" in foot; left foot swelled to double its size, and a small black spot like the prick of a pin appeared on the 2nd toe; this gradually increased in size, with acute pain	March 3rd.—Patient since admission has become restless and delirious; gangrenous patch on heel has spread markedly. 4th.—*Amputation through thigh.* Femoral artery found thrombosed. Patient's general condition became gradually worse in spite of the fact that the wound continued to do well. He became gradually exhausted, and died on the 10th	Death	Amputation through thigh; femoral thrombosis; no recurrence of gangrene; exhaustion.
Started 3 weeks ago with burning pain in right great toe, and tingling pain in sole of foot and up the calf of leg, with cramp in calf. This lasted till 10 days ago, when it began to diminish, and at the same time he noticed that skin over great toe was changing colour	Sept. 26th.—Discoloration of great toe has extended upwards about ¼ inch; patch on inner side of tip of 2nd toe; dusky area over 1st and 2nd metatarsal bones. Oct. 3rd.—Discoloration has extended to remaining toes, and area on dorsum has extended a little. 7th.—Great pain and sleeplessness; discoloration extending. 31st.—Line of demarcation forming over metatarso-phalangeal joints. Nov. 1st.—Patch on heel. Dec. 4th.—*Amputation through thigh*; long anterior flaps, commencing about 6 inches above knee; flaps very "anæmic," there being only one or two bleeding vessels; thrombus in femoral artery. 8th.—Pain in stump; good deal of sweet discharge from wound. Temp. 97° to 98° since operation. 26th.—A quantity of slough cut away from wound. Jan. 5th.—Patient became gradually weaker, and died	Death	Femoral thrombosis; amputation through thigh; sloughing of wound. P.M.—Ante-mortem clot in left heart; infarct in spleen; granular kidneys, 6½ oz.; commencing pneumonia on left side.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
41	M., 67	1889, 142	Feb. 21st, 1888. Well-nourished man. Urine contained albumen one tenth, and large quantity of sugar; anti-diabetic treatment	Left great toe gangrenous; sensibility greatly impaired in all the toes of left foot; foot and ankle œdematous; bullæ on 1st, 2nd, and 3rd toes
42	M., 62	1889, 144	March 13th, 1889. Patient very emaciated; radial arteries feel as hard as pipe-stems, quite incompressible, calcareous plates can be felt in them; urine contained large amount of sugar, no albumen; anti-diabetic treatment	All toes of left foot affected by dry gangrene; wide and deep line of demarcation over metatarso-phalangeal joints; foul smell
43	F., 67	1887, 130	April 13th, 1887. Thin and weak; rough systolic aortic bruit. Pulse irregular; vessels thickened	Dry gangrene of foot and lower half of leg; line of demarcation covered with granulations; redness and swelling of leg above this; small black patch over centre of knee; foul discharge
44	M.	1887, 131	Aug. 12th, 1887. Pulse hard; vessels thickened; temp. 102·2°. Urine contained a little albumen. Subconjunctival œdema	Gangrene extends up to middle of instep; beyond this foot is red and swollen; gangrenous great toe already separating; no pulsation in posterior tibial at ankle

Mode of onset.	Progress and treatment.	Result.	Remarks.
Commenced 14 days ago as a pimple or sore on great toe	*Amputation through thigh*, May 28th. No details given	Death, June 13th, 1888	Diabetes; amputation through thigh
About 13 months ago left foot began to swell and skin began to peel off; not much pain; attended Out-patients for 2 months, and foot became less swollen. A month ago he lost sensation in his toes, and a blister formed on great toe	March 16th.—Great deal of pain in foot. 18th.—*Circular amputation through middle of thigh*; femoral artery very much calcified; very little blood lost. 19th.—Pulse fairly good; has been very sick; temp. 96°. 20th.—Became very weak during night; breathing stertorous; pulse scarcely perceptible. Death	Death	Diabetes; arterio-sclerosis; amputation through thigh; coma.
Knocked leg a year ago. A spot of black appeared, which has been extending ever since	May 3rd.—*Amputation through thigh*; not much bleeding. 4th.—Patient bore operation well. Temp. 99·6°. 9th.—Considerable part of edges of flaps have united. June 2nd.—Healed everywhere but at site where drainage-tube entered. 22nd.—Went out well. Examination of leg.—Thrombosis of femoral artery at seat of amputation; clot granular, slightly adherent to vessel walls, not much decolourised. Beyond popliteal artery vessels were occupied by tough thrombus, which did not entirely occupy the lumen of vessels. Internal coat of arteries much thickened, representing half the thickness of the vessel wall; it could be readily dissected off. It was markedly atheromatous, and in some parts there were small calcareous plates. Separation of bone at line of demarcation was occurring as necrosed portion fell off during examination.	Recovery	Gangrene of foot and leg; amputation through thigh; good union.
No history obtained	Immediate amputation was advised. Aug. 12th.—*Amputation through thigh* by long anterior and short posterior flaps; posterior cut by transfixion; knife entered 6 inches above knee-joint; popliteal artery very rigid. Aug. 15th.—Stump had not been dressed, as no discharge had come through; no rise of temperature; fairly good night. Aug. 16th.—Patient died	Death	Chronic Bright's disease; arterio-sclerosis; amputation through thigh; exhaustion.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
45	M., 64	1887, 127	Admitted Feb. 8th. Urine 1025, acid, no albumen, much sugar; has been under treatment for diabetes for 5 years	—
46	M., 46	1886, 124	Admitted April 21st. Has always been a free liver, eating and drinking freely. Urine 1036, very acid; no sugar; considerable amount of albumen	Gangrene of 4th right toe; well-marked line of demarcation across centre of 1st phalanx; very offensive. Sole of foot inflamed and swollen; very feeble pulse in anterior and posterior tibial arteries

Mode of onset.	Progress and treatment.	Result.	Remarks.
—	Admitted Feb. 8th with Pott's fracture; put up in Croft. 14th. — Patient doing well; temp. 100·6°. 16th.—Over seat of fractured fibula leg is becoming black. 19th.—Lower two thirds of leg and foot are swollen and red, with numerous black patches scattered about; foot œdematous but not cold. 20th.—Two incisions made on outer side of crest of tibia into soft boggy area; blood and pus escaped. 24th.—Bone nearly projecting through on inner side; temperature subnormal. 25th.—*Amputation by method of Stokes-Gritti*; vessels very much diseased, enlarged, thickened, stood open like pipe-stems; coats calcareous. Anterior and posterior tibials obliterated below middle of leg; peroneal arteries very dilated and calcareous, but patent; muscular branches stood out as rigid tubes on cutting into muscles. 26th.—Recovered well from operation. Urine free from sugar, 1020; is not unduly thirsty; does not pass more than normal amount of urine. Tongue dirty white. 28th.—Sugar in urine. March 2nd.—Rather free sanguineous discharge from wound. Urine about 2 pints daily; no sugar; no albumen. 3rd.—Good union taking place in wound. Sugar did not reappear in urine till March 18th, from which time till April 18th it persisted in small amounts. After that time it contained a large amount. He was seen about the end of 1888; he was looking well, and was getting about on a peg	Recovery	Diabetes; Pott's fracture; arterio-sclerosis; gangrene; Stokes-Gritti amputation; marked diminution of sugar after amputation.
Commenced a fortnight ago as a swelling, the result of an inflamed soft corn, which broke and discharged; 4th toe subsequently became gangrenous	April 23rd.—*Toe amputated* at metatarso-phalangeal joint; tissues around were not at all healthy and did not give much hope of healing without sloughing. 27th.—Wound smelling foul; sole of foot much swollen and inflamed; two incisions made, with escape of foul chocolate-coloured pus. In the afternoon a black patch made its appearance on sole of foot. Violent hiccough for last 24 hours. *Amputation through lower part of thigh* by long anterior and short posterior flaps. Popliteal artery was extremely atheromatous, rigid, and open like a quill. Hiccoughing persisted. 28th.—Marked improvement in general condition; sweating less. Pulse 120; more perceptible. A good deal of sanguineous oozing through dressings; no signs of redness or gangrene in flaps. 29th.—Very weak; pulse 132, running; temp. 101·2°; hiccough and vomiting; flaps in good opposition; stump warm; no signs of gangrene. 30th.—Death	Death	(1) Amputation of toe; extension of gangrene. (2) Amputation through thigh; no recurrence of gangrene; fatty viscera; atheroma.

No.	Sex and age.	Year and volume.	General condition.	Condition calling for treatment.
47	M., 79	1883, 105	Admitted Dec. 14th, 1883. Body and limbs much wasted; 17 years ago had syphilitic ulceration of leg, which healed under treatment; has suffered frequently from cramps in calves; pulse 96; arteries hard and atheromatous; temp. 98·8°; tongue red and dry; always thirsty; urine 1039, small amount of albumen, sugar 23·32 grs. per oz.; anti-diabetic treatment	Left great toe black and shrivelled, with well-marked line of demarcation at base. Heads of 2nd and 4th metatarsal bones are black, dry, and rough; that of 3rd covered with granulations; sole of foot undermined for 1½ inches; 5th toe gangrenous, with line of demarcation at its base
48	M., 72	1883, 104	Admitted March 23rd. Has had several attacks of gout; hard drinker; temp. 101°. Much pain in foot; urine no albumen, no sugar; heart-sounds weak	Surface left by amputation of 5th toe is black; 4th toe is gangrenous, and from this a triangular patch of gangrene extends over dorsum of foot for about 3 inches; redness extending to above ankle; pain

Mode of onset.	Progress and treatment.	Result.	Remarks.
Last March noticed an ulcer on left foot between 1st and 2nd toes; foot was swollen at the time; ulceration spread to end of 2nd toe very slowly; 2 months ago 2nd toe was amputated; shortly afterwards 3rd and 4th toes died, and were amputated 6 weeks ago; 4 weeks ago 1st and 5th toes became gangrenous	17th.—7 pints urine in 24 hours, containing 3358 grs. of sugar. 23rd. — Extension of inflammation in foot; sugar 15·9 grs. per oz. 28th.—*Amputation through middle of thigh* by antero-posterior flaps; vessels much thickened, but not calcareous. 29th.—Death occurred somewhat suddenly about 12 hours after operation; skin perspiring; pulse 80, scarcely perceptible, and resp. 44, noisy shortly before death	Death	Diabetes. (1) Amputation of 3 toes in succession; recurrence of gangrene. (2) Amputation through thigh; ? diabetic coma; arterio-sclerosis. P.M. —Adherent pericardium; arteries thick walled; kidneys large and coarse.
In 1879 he had left great toe amputated for perforating ulcer into metatarso-phalangeal joint. 6 mos. ago a corn on right little toe became inflamed; 9 days ago toe was amputated for gangrene. Since then gangrene has been spreading gradually	Temp. varies between 99°—101°; the gangrene continued to spread, and by March 29th all the toes were black. 30th.—Line of demarcation forming. April 12th. —Tendons exposed; very offensive. May 2nd. — The slough does not separate very rapidly; he suffers great pain. 5th.—*Lisfranc's amputation* performed. There were no vessels required tying; wound continued to slough, and on June 8th a slough appeared on the heel. By July 16th all the bones in front of transverse tarsal joint had necrosed and come away. July 24th.—Patient very restless and wandering; temp. 99°. July 29th.—*Amputation at junction of middle and lower thirds of thigh* by antero-posterior flaps; subsequent suppuration. Aug. 15th.—Portion of skin over end of bone sloughed away. Oct. 31st.—2½ inches of femur having necrosed were removed. Dec. 16th.—Attack of pleurisy. Discharged Jan. 24th with slight discharge from two sinuses.	Recovery	Alcoholic; perforating ulcers. (1) Amputation of right 5th toe; spread of gangrene. (2) Lisfranc's amputation; spread of gangrene. (3) Amputation through thigh; sloughing; removal of 2½ inches of femur; slow healing.

ON

SCARLATINAL NEPHRITIS AND ITS VARIETIES.

By F. M. TURNER, M.D.

(Communicated by the Editors.)

In this paper I give the results of more than two years' study of this disease, which I have had extensive opportunity of observing at the North-Eastern Fever Hospital. During my stay there considerably over 6000 cases of scarlet fever have been treated, only a minority by myself; but of those under the care of my colleagues I have seen most that were severe or fatal, especially if complicated with nephritis.

Of the several interesting points which this disease presents the most interesting as well as most important is undoubtedly the capriciousness of its onset; and for this I can offer no explanation. There is no definite relation to the severity of the original fever, and none of the subsidiary causes as yet put forward are sufficient to explain the facts. Chills can hardly occur in hospital often enough to account for the large percentage of nephritis, especially as in the majority of cases this complication develops while the patient is in bed. A sudden change to damp weather, or a thaw in winter, I have occasionally found coincide with the appearance of several

cases of hæmaturia, but further experience has shown me that such is not always the case; and a steam kettle given for bronchitis or other cause has no influence in bringing on nephritis. Constipation may have some predisposing influence, but not much. Mahomed,[1] who assigned great importance to this, was forced to believe that one day's constipation was a sufficient cause. He, however, makes no statement as to how many times his patients passed over a period of constipation of that duration without ill effects; and in view of the fact that nephritis is often and probably generally well advanced before any clinical symptoms are apparent,[2] his ingenious theory becomes untenable. Some other supposed causes, as diet, overcrowding, &c., are mentioned further on in connection with my statistics.

Individual predisposition of course can only be assumed, not observed; but, if present, it certainly obeys no rules among members of the same family. Sometimes several brothers and sisters who have scarlet fever together will all develop nephritis; on the other hand, it often happens that of two brothers who suffer equally severely from scarlet fever, one develops nephritis and dies, while the other escapes altogether.

The question that next to this had the most interest to me was whether more than one pathological process occurred, —whether, in fact, the different course in different cases was merely one of degree; and whether the different appearances seen *post mortem* were to be explained as due to differences of stage reached at the time of death.

In 1883 Friedländer[3] published an account of three distinct varieties of scarlatinal kidney. During four years he made autopsies on 229 scarlet fever cases, and found signs of nephritis in about half of these. Three distinct forms occurred, which he described as follows:

1. Initial catarrhal, the early form.

[1] 'Med.-Chir. Trans.,' 1874, p. 197.

[2] See the following papers:—Klein, 'Reports of Med. Off. to Privy Council,' 1876 (n. s. viii), p. 24; Crooke, 'Fortschritte d. Medicin,' 1885, s. 651; Sörensen, 'Zeitschrift f. klin. Med.,' Bd. xviii, s. 298; Baginsky und Stamm, 'Arch. f. Kinderheilkunde,' 1893, Bd. xvi, s. 373; Willey, 'Med. Press and Circ.,' 1888, vol. ii, p. 644.

[3] 'Fortschritte d. Med.,' 1883, No. 3.

2. The large limp hæmorrhagic kidney, interstitial septic form.

3. Nephritis post-scarlatinosa, the glomerular form.

Transitional forms were totally absent, and mixed forms rare.

(1) appears with the exanthem, and does not prove fatal. It clears up after one or two weeks.

(2) also comes on early, but is very severe. It only occurred twelve times out of the 229. The kidney is enlarged and very limp; the marking of cortex has disappeared. The colouring is diffuse, and there are numerous hæmorrhages. Microscopically great extension of the interstitial tissue; little alteration of the epithelium. This form is connected with especially severe diphtheritic affections, cervical phlegmon, &c., and is probably only an indirect consequence of scarlet fever.

(3) is an affection characteristic of scarlet fever. The kidney may or may not be hyperæmic, is firm and normal except that the glomeruli project everywhere as grey points above the cut surface. Under the microscope they are almost completely bloodless, and show an extreme overgrowth of nuclei, and are much enlarged. The capillary loops are changed to solid masses. This leads to œdema, hypertrophied heart, suppression of urine, and death. Forty-two cases out of the 229 showed this change.

The glomerulo-nephritis was first described by Klebs,[1] who regarded it as characteristic of scarlet fever, though he states that other forms of nephritis may also occur in this disease.

No two authors are exactly agreed on the subdivisions of acute nephritis, whether dealing with scarlatinal forms only or all forms together; but I have given Friedländer's descriptions at some length, since I find them to apply very well to my own cases. In fact, I had collected the bulk of my pathological material before becoming aware of his work, and had independently come to the conclusion that two types of fatal nephritis occurred, corresponding to his septic and glomerular forms.

[1] 'Handbuch d. pathologische Anatomie,' 1869, s. 644.

Sörensen[1] has paid special attention to this question, and to the correlation of the clinical history with the *post-mortem* appearances. He finds no evidence of two distinct processes, but thinks that all the cases form a series of which the early and most severe are of interstitial, whilst the late and more insidious are of glomerular type.

I agree with him against Friedländer that mixed forms are frequent, but not so frequent as the extremes; I hold, however, that the latter are so distinct as to require the supposition of two distinct causes, to the occasional combination of which the intermediate forms are due.

It is often possible during life to diagnose one or other of these two forms, but as regards the other lesions which I have found, not coming under these two types, I have not been able to correlate them with any well-defined clinical course or set of symptoms.

As regards the post-scarlatinal cases, when complete blocking of all or most of the glomeruli has taken place the cause of death is obvious, but in some of my cases this change has not taken place. In these, though slight pathological changes may be present, I have not been able to satisfy myself that they are more advanced than in many kidneys which have undoubtedly not been the cause of death.

On account of the want of correspondence between the clinical course and the *post-mortem* phenomena, I will separate my remarks on these two heads, giving, however, the connection between the two wherever possible. For statistical purposes, however, I have compiled a list of fatal cases, including all in which, either on clinical or *post-mortem* evidence, death could be ascribed either wholly or in greater part to kidney disease.

I. Statistical.

The proportion of scarlet fever cases which develop nephritis has been very variously given by different observers, and it is usually believed to vary with different epidemics, and also with the surroundings and treatment of the patients.

[1] 'Zeitschrift f. klin. Med.,' 1891, Bd. xviii, s. 298.

In the statistical reports published by the Metropolitan Asylums Board albuminuria and nephritis are tabulated as distinct complications of scarlet fever. This distinction, if insisted on, is sufficient to upset all statistical comparisons, for although many cases present albuminuria without other evidence of nephritis, there occur all grades from the former class up to severe and fatal nephritis. That any distinction between the two classes is purely one of degree I am from my own observations quite convinced. It follows that the line of distinction must be quite an arbitrary one, and it will naturally vary with different observers. A comparison of the figures given in the above-mentioned reports for different hospitals will bear out this conclusion; thus in 1893 the albuminuria cases at one hospital outnumbered the nephritis cases by four to one, at other hospitals they were less than one to one.

It is of course possible to assume that though the mildest cases of nephritis may show no symptom beyond albuminuria, yet many cases of albuminuria are not due to mild nephritis. I can see no grounds for this supposition, and even if true the desirability of uniting the two classes for statistical purposes is not affected.

During the two years 1893–4, 5109 cases of scarlet fever were completed at the North-Eastern Hospital, and of these 673 = 13·1 per cent. had albuminuria or nephritis. This number does not include any cases where albumen was detected on less than three successive days, and possibly some other slight cases are omitted. Taking all the hospitals under the Asylums Board during the year 1893, the incidence was practically the same, viz. 12·9 per cent., but I cannot say whether a uniform practice prevailed as to the omission of the most transient cases. In the years 1888 to 1892 the ratio varied from 20·5 per cent. to 11·7 per cent.

TABLE I.—*Cases at the North-Eastern Hospital. A large number of these came under my personal observation.*

Year.	Scarlet fever. No. of completed cases.	Albuminuria.	Nephritis.	Total.	Percentage.
1893	3005	192	201	393	13·1
1894	2104	135	145	280	13·3
Total .	5109	327	346	673	13·1

TABLE II.—*Cases at all the Asylums Board Hospitals. Drawn from the Annual Reports.*

Year.	Scarlet fever. No. of completed cases.	Albuminuria.	Nephritis.	Total.	Percentage.
1888	5,717	867	223	1,090	19·0
1889	3,744	598	171	769	20·5
1890	6,439	609	149	758	11·7
1891	5,444	587	200	787	14·4
1892	11,326	1,470	431	1,901	16·8
1893	14,867	1,197	719	1,916	12·9
Total .	47,537	5,328	1,893	7,221	15·2

Other observers give very different figures. Goodall [1] out of 5443 scarlet fever cases found 476 (= 8·74 per cent.) with nephritis. In the latter class he includes all cases where albumen is detected on more than one occasion, except when obviously due to some other cause, as leucorrhœa, menstruation, or the febrile condition. With his omissions I certainly agree except as to "the febrile condition." I shall show below that some severe cases of nephritis are apt to be diagnosed "febrile albuminuria" until a post-mortem is performed. Caiger [2] out of 4015 cases found simple albuminuria or nephritis in 7·69 per cent., excluding cases where albumen is observed on less than three successive days. On the other hand, some observers record a much larger percentage than my own.

[1] 'Guy's Hosp. Rep.,' vol. xlvi, p. 91.

[2] 'St. Thomas's Hosp. Rep.,' 1893, p. 109.

Sörensen,[1] out of 365 cases, found 73 (= 20 per cent.). Of these he distinguishes four grades, viz. abortive, sixteen cases; slight, forty-two cases; severe, eight cases; very severe, two cases. Of the remaining five he gives no account. Dudley[2] found forty-nine cases of albuminuria out of 100, testing the urine of each case daily. Of these only a few had any other evidence of nephritis, but many had otitis. Greswell[3] found albumen in 316 cases out of 347. These latter cases are all included in the statistics of Table II. Thompson,[4] whose work is I believe the most careful of any that has yet been done on the subject, found 112 cases out of 180 = 62·2 per cent.

My own impression is that the rate of incidence of albuminuria varies a great deal at different times, especially if only small numbers are dealt with; but so much depends on the frequency of testing, the particular test relied on, the brightness of illumination, as well as on the observer, that the results of different observers are not in the least comparable. Thus Thompson, at Glasgow, with only 180 cases in the course of a year, was able to devote himself to a most painstaking research, testing a specimen from each patient whenever possible three times a day, and even going so far as to concentrate specimens in the cold in cases of doubt. In my own cases, and in those of the other medical officers of the Asylums Board, the much larger number treated rendered such elaboration impossible, testing being done only on alternate days, and probably more hurriedly; and I think the difference in methods quite suffices to account for the wide difference of result.

Unfortunately this incomparability of different statistics makes it impossible to judge truly the effect of treatment on the appearance of albuminuria, but if one relies on them, one finds no ground for the prevalent belief in strict farinaceous diet with confinement to bed as a preventive. Thus the Glasgow cases, of whom 62 per cent. had albumen, were all confined to bed for four weeks, and to the ward another

[1] Loc. cit.

[2] 'Jahresberichte d. ges. Med.,' 1892.

[3] Ibid., 1890.

[4] 'Med.-Chir. Trans.,' 1886, p. 97.

week; while the diet was farinaceous for two to three weeks, and in thirty cases till the middle of the fifth week. The Asylums Board cases were not so strictly treated. I know that at several hospitals three weeks in bed is the usual routine, and patients are allowed out of doors almost as soon as they get up, provided the weather is suitable. To leave statistics, and speaking from the general impression gained in my own experience, I find the same result. I have differed from most of my colleagues at the North-Eastern Hospital in never enforcing a routine of three weeks in bed, and I usually get my patients up in a fortnight if convalescent, without being able to detect any difference in the prevalence of albuminuria or nephritis.

The number of fatal cases of nephritis is not so liable to statistical fallacies as the number of nephritis alone. Out of the 5109 cases given in Table I fifty-five died with nephritis, either alone or complicated with other lesions. This number includes those who presented severe symptoms during life, as well as some others in whom, post mortem, extensive disorganisation of the kidney was found, though during life there were no sufficient symptoms for diagnosing such a complication. This gives a death-rate of 1·07 per cent. This number may possibly be too low, since three or four patients died with septic conditions and slight amounts of albuminuria on whom no post-mortem examination was made. I strongly suspect that some of them would have shown the large septic kidney if examined. Goodall found from about the same number of cases a death-rate of ·6 per cent. The difference is partly due to the greater average age of Goodall's patients, among whom were included an abnormal number of adults.

The following table shows the age and sex mortality from nephritis. Unfortunately the ages of the completed scarlet fever cases are not given in the statistical report, but only the ages of cases admitted during 1893 and 1894. However, the totals only differ by 150, and may be considered approximately equivalent.

TABLE III.—*Showing the number of cases of fatal nephritis at each age period, and of each sex, occurring at the North-Eastern Hospital in* 1893–4, *with the death-rates at each period.*

Year.	Nephritis deaths.			Scarlet fever.		Percentage mortality from nephritis.
	Male.	Female.	Total.	Admissions.	Discharges and deaths.	
Under 1	1	0	1	—	These numbers are unfortunately not available	—
1—2	4	2	6	—		—
2—3	2	3	5	—		—
3—4	6	2	8	—		—
4—5	9	2	11	—		—
Total under 5	22	9	31	1433		2·1
5—10	9	9	18	2057		·9
10—15	4	1	5	945		·5
Over 15	0	1	1	520		·2
Total .	35	20	55	4955	5109	1·07

Sex.	Nephritis deaths.	Scarlet fever.		Percentage mortality from nephritis.
		Admissions.	Discharges and deaths.	
Male . . .	35	2539	—	1·4
Female . . .	20	2416	—	·8
Total . . .	55	4955	5109	1·07

It will be seen that the disease is more fatal to males than females, and more fatal the younger the patient. The high mortality among children is, however, not so marked as in scarlet fever itself, including deaths from all complications. Thus the table shows a mortality under five years of age double that of all ages together, and not much more than double that of the second quinquennium ; whereas the statistics of the Asylums Board show for scarlet fever itself a death-rate under five years more than double the total rate, and more than treble the death-rate between five and ten years.

The following tables give various statistical information concerning the before-mentioned fifty-five fatal cases.

TABLE IV.—*Date of onset of nephritis in relation to the stage of scarlet fever.*

During acute illness	23
During convalescence	22
Intermediate cases in which the temperature sank to normal only one or two days before the symptoms of nephritis were noted	7
Date of onset doubtful	3
	55

The date of onset is taken as the date on which the first symptom, usually albuminuria, less often sudden rise of temperature, appeared.

TABLE V.—*Date of death reckoned from the onset of scarlet fever.*

8th day . . . 1	25th day . . . 3	36th day . . . 4
14th „ . . . 1	27th „ . . . 2	38th „ . . . 1
17th „ . . . 1	28th „ . . . 1	40th „ . . . 1
18th „ . . . 1	29th „ . . . 3	41st „ . . . 1
19th „ . . . 2	30th „ . . . 3	42nd „ . . . 1
20th „ . . . 1	31st „ . . . 1	47th „ . . . 1
21st „ . . . 2	32nd „ . . . 4	64th „ . . . 1
22nd „ . . . 2	33rd „ . . . 3	Doubtful . . . 2
23rd „ . . . 1	34th „ . . . 2	55
24th „ . . . 7	35th „ . . . 2	

It will be seen from this table that the majority of deaths took place between the twentieth and fortieth days, later than which the deaths are very few. It is well known, however, that deaths may occur from this disease after months or years, so that the remarkable falling off in the later period of the above table seems to require explanation. The fact is that in nearly all the cases the disease showed severe symptoms from or shortly after the commencement, and from this acute form the patient either died in a short period, rarely exceeding three weeks, or else recovered. In a certain number of cases albuminuria persisted for a long time, but mostly in very slight amount, and some of these

patients were sent home in this condition, but only after all symptoms had disappeared, and in most cases having been up and running about for some weeks. Such cases are probably liable to acute exacerbation after they reach home, and patients also who have passed two or three months from the onset of fever have been known to contract nephritis; but apart from this risk the nephritis patients who are discharged uncured appear quite healthy.

TABLE VI.—*Dates of death of nephritis cases compared with the number of scarlet fever cases in hospital.*

Month.	1893.		1894.	
	Nephritis deaths.	Max. number of cases in hospital.	Nephritis deaths.	Max. number of cases in hospital.
January	3	445	1	429
February	4	517	0	344
March	3	566	0	283
April	1	503	7	292
May	0	488	9	275
June	4	543	4	281
July	3	510	3	300
August	2	502	0	322
September	1	501	0	345
October	0	441	5	386
November	2	494	0	382
December	3	500	0	320
Total	26	—	29	—

For a rough comparison I have given the maximum number of patients under treatment occurring in each month. This is sufficient to show that crowding of patients had no tendency to increase the nephritis death-rate. On the contrary, during 1893 only 26 deaths occurred from that cause out of 3005 patients, whereas in 1894 29 cases occurred out of 2109. During the former year all the wards were often obliged to contain 30 patients for long periods together, although originally designed for twenty only.

Goodall, with an equally large number of patients, found that crowding had no effect on the incidence rate of

nephritis; whereas Sweeting,[1] with more limited material, found that it caused a large rise.

The table does not show any seasonal prevalence, the rises in one year almost exactly corresponding to falls in the next. The extreme nephritis mortality during the spring of 1894 coincided with a mortality from other causes, chiefly severe affections of the throat and septic lesions, far in excess of the usual rate at this hospital.

II. Clinical.

I shall not attempt a complete clinical account of this disease, but will confine my attention to those points wherein I think the accounts of previous writers either deficient or mistaken. In most treatises on medicine scarlatinal nephritis is taken as the type of acute Bright's disease, yet the descriptions given differ markedly from what I regard as the normal course of the disease. Great prominence is usually assigned to dropsy as a characteristic symptom, or even the earliest symptom. Cases in which dropsy is absent are regarded as quite exceptional. I need only quote the following.

Saundby ('Lectures on Bright's Disease,' p. 175): "Œdema . . . is less constant than has been stated. It is notably absent in the majority of cases [of acute nephritis] occurring in the course of acute diseases. The great exception to this rule is scarlatina."

Henoch ('Lectures on Children's Diseases'[2]): "There may be no œdema whatever during the whole course of the disease; but in most cases œdema is observed sooner or later."

F. Taylor ('Practice of Medicine'[3]): "In the more typical cases the first symptom is dropsy."

Many writers, as Barlow and Goodhart ('Guy's Hospital Reports,' vols. v and xxxix), use the terms scarlatinal dropsy and anasarca after scarlet fever, and seem to ignore cases of scarlatinal nephritis without dropsy. As indicating the prevalence of the view that dropsy is characteristic, I may mention that several observers in publishing cases in whom

[1] 'Metropolitan Asylums Board Rep.,' 1887.

[2] New Sydenham Society's Translation, vol. ii, p. 140.

[3] Third edit., p. 729.

no dropsy was found remark on its absence as a curiosity (Biermer, 'Virch. Arch.,' xix, s. 537; Willey, 'Med. Press,' 1888, vol. ii, p. 644).

My own experience is entirely opposed to the view that dropsy is a characteristic symptom. Speaking for the present only of those cases which are treated in hospital from the beginning, I may state that marked œdema never occurs. A slight puffiness of the face is not uncommon, but with fat and chubby children one may be easily deceived, and where present the swelling affects the cheeks rather than the eyelids, where its presence would be less open to the above doubt. Œdema of other parts—ankles, loins, and scrotum—is absent in the majority of cases. Œdema of the dorsum of foot and hand is not particularly rare in the exanthem stage, but is much rarer with nephritis. In what proportion of cases it might be found in any of these situations by careful examination I am unable to say, as a short experience is sufficient to convince one of the exceptional nature of its occurrence (and therefore to take less pains in observing it), though I have from time to time examined for it in all my cases of well-marked nephritis, and in the majority have found it absent.

The following case is the only one in which I can remember to have seen extensive œdema in a patient who developed nephritis within the hospital. In this case both heart and lungs showed considerable damage before the nephritis developed, and it would be wrong to attribute the œdema to the nephritis alone.

Case 1.—M. Z—, female, æt. 7.

November 7th, 1894.—Vomiting, sore throat.

9th.—Rash.

10th.—Admitted. Throat very foul. Drowsy.

16th.—Temperature varies from 38° to 39° C.; livid; no râles in chest.

26th.—Pyrexia and lividity continue; fauces clean, but now the pharynx is seen to be covered with exudation; no albumen in urine; bronchitis.

28th.—Trace of albumen; dulness at base of left lung.

December 1st.—Pitting over both feet; pharynx still very dirty; temp. 37° to 39°; pulse rapid, weak; still cyanosed.

5th.—More œdema; legs pit easily as high as knees; the arm on which she lies is very swollen; profuse hæmaturia.

15th.—Breathing more rapid; dulness, &c., in left axilla. Exploring needle used, turbid fluid found containing many streptococci.

16th.—Incision and drainage, several ounces escaped; less œdema of legs. The temperature returned to normal almost at once, and the urine improved rapidly. A faint trace of albumen, however, could be detected on 25th February, when she was discharged.

Previous writers with fever hospital experience take much the same view as myself, but state the case as to absence of œdema rather less strongly. Thus Goodall,[1] at the London Fever Hospital, concludes that "marked anasarca is unusual except in neglected cases." Caiger[1] at the South-Western Hospital also states that "anasarca to any extent is rare." But Thompson,[1] of Glasgow Fever Hospital, found it present in 24 cases, two of whom had no albumen; the rest occurred among 112 albuminuric patients. In 12 it was considerable in amount.

In great contrast to these cases are those of which perhaps a dozen have come under my observation at the hospital, who develop dropsy while at home.

Case 2.—C. J. E—, male, æt. 11.

May 21st, 1893.—Vomiting.

June 2nd.—Admitted; distended abdomen; much œdema of loins, scrotum, and legs; albuminuria; profuse desquamation. Numerous raised erythema patches on abdomen and legs, some hæmorrhagic; some also on arms. Treated by hot baths, poultices to loins and aperients, and later on acetate of ammonia and iron. The former produced good perspiration.

15th.—Œdema less.

September 15th.—No trace of œdema.

27th.—Discharged; still passing a small amount of albumen. The temperature was normal throughout, as was also the amount of urine passed. In this case the scarlet rash was never observed, but there was no doubt that the attack was post-scarlatinal.

The exact cause of the difference in these two sets of cases is not known, but there is no doubt that under hospital conditions œdema is rare, while under other conditions it may be extreme. Probably the difference is due to some special cause, such as exertion, the erect posture, or possibly chill.

The hospital routine which is so successful in avoiding

[1] Loc. cit.

the onset of œdema is of the simplest. Neither rest in bed nor a vegetarian diet is enforced so long as at many institutions, my own patients as a rule having been allowed fish and one egg daily as soon as the temperature has reached normal, and been allowed up at the end of two weeks. At the first onset, however, of albuminuria the patient is usually put back to bed and to farinaceous diet, but in many of my cases where only traces of albumen are discoverable I have not enforced either precaution.

Many observers have tried to distinguish scarlatinal albuminuria from scarlatinal nephritis, though some admit that the distinction is a purely arbitrary one of degree. If they lay any stress on œdema as a distinguishing character, a very false division will result. I think Baginsky has fallen into this error ('Arch. f. Kind.,' 1893) when he states that the hospital routine almost prevents nephritis, at least its severer forms. Out of 195 scarlet fever cases he had no deaths from nephritis excluding those admitted with it, and no cases developed severe œdema. That he had no fatal cases among that number is not surprising, as we have several times had as good a record at the North-Eastern Hospital, even without excluding the cases who have developed the disease before admission. The absence of severe œdema is only the natural course of the disease, and that its presence or the reverse is no test of the extent of damage to the kidney the fatal cases fully prove.

The temperature is usually raised at the onset in all but very slight cases, but in a minority considerable albuminuria may occur, and last a long time without pyrexia. Such cases, as a rule, show no symptoms except pallor. The most typical course is to get a sudden rise, often to 40° C., with a sudden fall, sometimes back to normal, sometimes not, after which sudden variations occur for a few days without any regularity. Often these sudden rises are accompanied by profuse sweating, but the skin is sometimes quite dry. In one or two cases only I have seen a rigor. After a few days the temperature, even in severe cases, approaches normal, and may continue so till death, but in a few cases continued high fever lasts days or weeks. Hyperpyrexia occurs before death in a considerable proportion of cases.

As regards perspiration, it is generally present at the onset, and often profuse, but if not spontaneous, may easily be produced by treatment. After a week or more there seems less tendency to perspire, and hot baths, drugs, &c., act less readily.

Lumbar pain I have not met with, even in children whom one would suppose to be old enough to complain of it, nor have I found tenderness on pressure in that region.

In cases with a sudden onset vomiting once or oftener is the rule; should the vomiting continue the prognosis is very grave. I have seen very few recover after obstinate vomiting, even though the obstinacy was not so great but that treatment has in time succeeded in checking it. Out of the fifty-five fatal cases no less than forty-seven showed this symptom,—nineteen, however, only slightly.

The urine on the day of onset shows a smoky colour with a deposit of a deeper beef-tea-like colour. In the deposit, blood, epithelial and granular casts abound. The proportion of blood usually diminishes, but that of albumen increases. The amount of urine passed daily becomes reduced to a few ounces, but often increases after a time. Total suppression for twenty-four hours is uncommon even in fatal cases.

The usual course of a fatal case is as follows. The onset is commonly sudden with vomiting and pyrexia, either during a prolonged initial illness or during convalescence. If the patient is old enough, he will probably complain of headache. The face becomes pale at once or in a few days. The pulse is rapid, of low tension, and often irregular. Sometimes there is an apical systolic bruit. Displacement of the apex-beat a little outwards is common, but I have seen no marked dilatation or hypertrophy post mortem. Drowsiness soon sets in, and is generally a marked feature till near the end, when it is succeeded by constant restless tossing about. Meanwhile vomiting may occur after everything taken, even water alone; emaciation follows. The patient is semi-comatose, passing urine and fæces in bed. The tongue gets dry and coated with sordes. Bleeding from lips or nose is common. For the last day or two the restlessness above mentioned sets in; the patient is continually turning over, or even tries to

get out of bed; every movement is very tremulous. His constant muttering, owing to the muscular weakness and the dryness of mouth and throat, is quite unintelligible, though I believe that partial consciousness is retained nearly to the end. In some cases convulsions set in shortly before death, but several such cases have shown purulent meningitis post mortem.[1]

In many cases a bright-coloured erythema comes out a day or two before death, somewhat resembling measles in general aspect, but the patches are brighter and more distinct in outline. It may affect the limbs only or be universal. Of the 55 cases, 19 presented a rash, usually of this character. In 4 it occurred on the day of death, in 1 the day before, in 5 two days, in 3 three days, in 1 four days, in 2 six days, and in 3 over six days before death.

Other modes of death are less frequent. Six of the above cases died suddenly; two of them dropped back while sitting up in bed, one of whom had only had albuminuria since the day before, though advanced septic nephritis and infarcts were found post mortem; one, who was admitted with much œdema on the eighth day of the scarlet fever, died the same day, apparently in a fit; one had severe dyspnœa and collapse due to cardiac dilatation, and fatal in a few hours—this is the Case 10 described below as typical glomerular nephritis—one had hyperpyrexia, and the remaining one had a curious set of symptoms,—collapse, sweating, and drowsiness, accompanied by closure of the air-passages every time he commenced to sleep. No post-mortem was allowed.

In an important class of cases the symptoms are almost or entirely latent.

Case 3.—Algie B—, male, æt. 5.

September 27th.—Onset.

28th.—Admitted. Temp. 39·4° C.; scarlet rash; fauces dirty; large cervical glands.

October 3rd.—Glands larger; temperature oscillates about 39° C.

5th.—Temp. 39° to 40° C.; redness and œdema of skin over the enlarged glands behind the sterno-mastoid.

[1] In view of a possible connection between purulent meningitis and acute nephritis compare three cases given by Crooke and Nason in 'Lancet,' 1889, vol. ii, p. 1217.

6th.—Skin angry below clavicle.

7th.—Cloud of albumen; flatulence; cervical inflammation apparently subsiding; on incision below the clavicle a thin watery fluid was found beneath the deep fascia; no abscess cavity.

9th.—Only watery discharge; less thickening around wound; liver and spleen enlarged.

10th.—Pleuritic rub on right side.

13th.—Signs of solidification of anterior margin of left lower lobe; death; temperature high throughout. Urine in small amounts at the last, but passed in bed.

The diagnosis made during life was pyæmia with visceral abscesses, but post mortem there was found extensive purulent infiltration of the sterno-mastoid muscle and the surrounding glands, a little pneumonia, early nutmeg liver, and enormously enlarged kidneys, weighing 20¾ ounces.

In these cases the gravity of the septic symptoms may mask the nephritis, however severe. The urine is often passed in fair quantity, and does not contain much albumen, and usually no blood. The albuminuria is therefore often thought to be merely febrile until the post-mortem is made.

This form of kidney may, however, show more signs of its presence, especially vomiting, less commonly marked diminution of the secretion. One case had suppression for twenty-four hours some days before death, followed by secretion of an almost normal quantity.

Sometimes the cerebral symptoms are so marked as to lead to a diagnosis of meningitis or cerebellar abscess.

Case 4.—E. B—, male, æt. 4.

April 19th.—Onset of scarlet fever.

23rd.—Admitted. Temp. 38·4° C.; small scalp wound.

24th.—Temp. 40·4°.

May 8th.—Temperature has never been below 38°; trace of albumen.

10th.—Vomiting; cloud of albumen.

11th.—More vomiting; passes urine freely in bed.

12th.—Temperature normal.

14th.—Refuses food; double otorrhœa; restless.

16th.—Enlarged liver and spleen; distended abdomen; squint; head retraction; headache; sighing respiration.

17th.—Vivid blotchy rash; death. *Post mortem*, excess of cerebral fluid, two ounces being collected; part of base of heart extensively hæmorrhagic, and covered with loose irregular lymph. A little fatty degeneration in the columnæ carneæ; old adhesions of lungs; two infarcts in spleen; kidneys 15½ ounces, surface pale; on section cortex much thickened, one infarct seen.

These cases all belong to the class of septic nephritis of Friedländer, and are associated with severe septic conditions, but cases of post-scarlatinal nephritis are also often associated with suppuration or less severe inflammations. Thus it is quite common during convalescence from scarlet fever for hæmaturia to occur simultaneously with sudden enlargement of the cervical glands, or to find that one precedes the other by a few days only. This coincidence is noted both by Goodall and Caiger. In many cases suppuration of the glands occurs, and after incision the hæmaturia clears up.

Case 5.—M. N—, female, æt. 5.

February 13th, 1895.—Onset of scarlet fever.

14th.—Admitted with slight rash and slightly inflamed fauces.

15th.—Slightly enlarged glands on right side of neck.

18th.—Temperature normal, and remained so until March 4th.

March 1st.—Glands still large; trace of albumen in urine.

5th.—Complains of throat, which looks red; temp. 38·2° C.; urine smoky.

6th.—Glands larger; more albumen; temp. 39·6°; skin hot, moist.

7th.—Hot bath to be given daily. Urine, 10 to 15 ounces in the day.

14th.—Slight pitting on shins, with considerable pressure; is very thin, face rather swollen, pale; drowsy; temperature usually between 37° and 38°.

23rd.—More lively.

25th.—Temp. 40·6°; vomited.

26th.—Headache; pulse rapid, low tension; urine dark with blood.

27th.—Temperature normal; pulse irregular; still pale, not drowsy.

April 5th.—Gland on left side much larger, hard and tender; poultice to neck; temp. 39·2°; more blood and albumen.

6th.—Fluctuation; incision, much pus escaped; after this a rapid decrease of the albuminuria.

I have several times seen alveolar abscess accompanied with hæmaturia in the same manner, while the same has occurred with the greater number of mastoid abscesses which have been operated on at this hospital.

Case 6.—F. B—, male, æt. 10.

February 25th, 1895.—Onset of scarlet fever.

26th.—Admitted with profuse rash, fauces deep red, and showing some deposit. Temp. 38·8° C.

March 3rd.—Temperature normal.

9th.—Temp. 37·8°; no albumen.

11th.—Temp. 38·4°; faint trace of albumen.

12th.—Large brawny swelling attached to right side of lower jaw; a carious molar tooth removed.

14th.—Swelling very large, and skin over it tense; it pointed in floor of

mouth; under gas an opening made, letting out a little broken-down tissue, no fluid pus.

17th.—Less swelling; still albumen; temperature normal.

18th.—Oleate of mercury applied.

22nd.—Swelling smaller but very hard; an enlarged gland just behind it; temp. 39·8°.

23rd.—Temperature normal; hæmaturia; ointment omitted.

26th.—Another rise of temperature; urine gradually improved, and was free from albumen on April 3rd.

Case 7.—L. R—, female, æt. 7.

January 23rd.—Onset; admitted; temp. 39·6° C.; bright rash.

29th.—Left otorrhœa.

February 1st.—Temp. 37·8°.

7th.—Temp. 38·6°; no albumen.

8th.—Cloud of albumen.

9th.—Hæmaturia.

10th.—Temp. 39·2°; albumen $\frac{1}{12}$; no œdema of feet, loins, or face; no lumbar tenderness.

13th.—Headache, nausea; sallow complexion; poultice to loins.

16th.—Slight œdema of eyelids and over sacrum, none on legs; heart irregular; albumen $\frac{1}{3}$.

17th.—Fluctuating swelling behind left ear; right otorrhœa.

18th.—Incision letting out some subperiosteal pus; gouging into mastoid antrum.

19th.—Less œdema of eyes, none of sacrum; temperature falling; albumen $\frac{1}{8}$.

March 3rd.—Sudden rise of temperature to 40·4°; headache; vomiting; wound healing; optic discs normal; albumen has very much decreased, but there is still a little blood.

4th.—Rather more blood and albumen; sweating.

5th.—Temperature normal.

16th.—No albumen.

22nd.—Got up.

April 9th to 11th.—Slight trace of albumen; wound not quite healed.

Case 8.—M. S—, female, æt. 10.

October 8th.—Onset of scarlet fever.

10th.—Admitted; foul throat; temp. 39·8° C.

11th.—Temp. 40·4°.

12th.—Cloud of albumen.

14th.—No albumen; temp. 37·8°.

26th.—Temperature approximately normal for four days; very drowsy, perhaps deaf; double otorrhœa.

28th.—Trace of albumen.

November 3rd.—Temp. 38·6°.

4th.—Right mastoid abscess; thick cloud of albumen.

5th.—Much œdema of right eye, and spreading across the nose to the left eye; no œdema elsewhere.

6th.—Incision, gouging into antrum.

7th.—Œdema of left eye only. She always lies on the left side.

8th.—Trace of albumen; temperature normal; much brighter in spirits; no œdema.

December 5th.—No albumen.

Of the cases where I have found the kidneys post mortem to be free from interstitial change only one had an abscess, but many had adenitis coming on about the same time as the nephritis.

The connection between nephritis of any form and suppurative or septic processes is so close, that in all cases where the advisability of operation is in doubt I regard hæmaturia or other well-marked signs of nephritis as an indication of urgency. Thus in Case 1 (M. Z—) the pleuritic effusion was by no means purulent, only turbid, with a few flakes of lymph, and of itself might have justified waiting, but the presence of hæmaturia, together with numerous streptococci in the fluid withdrawn, determined me to operate, with perfectly successful result, not only as to the pleura, but also as to the kidney.

The relation of the septic process to the nephritis is uncertain. In many of the cases of septic nephritis the former is obviously the cause of the latter, and I am inclined to think that this holds also with many of the glomerular forms. On the other hand, glandular swellings of the neck seem an insignificant cause for serious attacks of nephritis, especially as similar swellings frequently occur after scarlet fever without any such result. I believe that many such cases, especially those where the glands and kidneys both appear to be attacked at once, are due to a simultaneous attack by some virus, either living or chemical.

It is well known that subjects of Bright's disease are liable to septic inflammations following wounds, but this observation has been made chiefly on subacute and chronic cases, in many of whom œdema is a predisposing cause, and that it applies to acute cases in the first few days or weeks I have seen no evidence. On the contrary, operation wounds, mostly incisions to let out pus or relieve tension, usually do very well.

III. Morbid Anatomy and Histology.

I shall first describe and give typical examples of the septic and glomerular nephritis, and then those cases which do not fit either type. Unfortunately much of my material was obtained when I was very busy, and has often suffered from faulty preparation.

The material was hardened in Müller's fluid or spirit, in one or two instances only in Flemming's fluid. For examining the tubules I prefer cutting on a freezing microtome, as the sections stain better, and the paraffin method introduces further risk of distortion. But for the glomeruli, especially if diseased, I find paraffin embedding essential. Most sections I have stained with logwood and eosin, but for showing the basement membranes I find picro-nigrosin useful. In the glomeruli it stains the membrane of the capillary wall, and often shows clearly the distinction of epithelium from the endothelium and capillary contents.

1. *The septic kidney.*—A very good example is from Case 3 (Algie B—), whose clinical history is given above.

Both kidneys enormously enlarged, weighing together 20¾ oz. Capsule strips easily causing some laceration, since the cortex is very friable. Externally mottled, colour fairly dark. On section cortex much enlarged, deeply congested, a good deal stained with broken-down blood. Striations mostly visible, but there are many isolated white specks. Medulla darker than cortex, in part showing structure well, elsewhere irregularly stained, also showing some white specks. At junction of the two zones a very well-marked irregular white band, varying in thickness. It showed in places a tendency to break up into crowds of small white foci, which scattered away from it into either zone. A few petechiæ in the pelvic mucous membrane.

Microscopically a very extensive round-celled infiltration, which over more than half the cortex leaves no normal structure visible. The infiltration is densest round the larger vessels, also round the glomeruli. The latter show all their loops distended, and with thin walls. In the less intensely affected areas the basement membranes only of the tubules are visible, and the lumen is crowded with cells of various sizes. The healthy tubules are often stuffed with casts or show a dilated lumen.

The boundary zone shows the most intense cell infiltration, and the medulla is also affected, but to a less extent.

The normal weight for a boy of his age (five years) would be about four ounces, so that a weight of five times the

normal was reached. Although so little of the secreting epithelium was left the diminution of the excretion was not marked, but measurement of the amount was not possible.

In no other case have I seen so great an enlargement, but in many the weight was twice, three times, or more than the normal. But by microscopic examination two cases showed still more extensive infiltration of round cells, so that only in very few places are any secreting tubes visible. In one of these suppression for twenty-four hours occurred five days before death, but after the re-establishment of secretion urine was passed often and in fair quantity until death, though here also accurate measurements could not be made (Case 9, W. W—).

The characteristics of this type, which are well shown in all my cases, are the extreme enlargement, chiefly affecting the cortex, the limp consistency and friability, the loss of structure markings, sometimes complete, sometimes not, and their replacement by small inflammatory foci; and to microscopic examination the destruction of at least half the cortex by round cells, and the distension of the glomerular capillary loops.

In only one case have I seen many hæmorrhages, and in that the kidney was spotted with raised red areas on the external surface. On section these were seen to extend diffusely in the cortical substance.

Well-marked examples of this type have been described by most authors—Aufrecht,[1] Baginsky and Stamm,[2] Biermer,[3] Coats,[4] Crooke,[5] v. Kahlden,[6] and Wagner.[7] Several of Klein's[8] cases were similar, but less marked.

2. *Glomerular nephritis.*—I have only one specimen which corresponds exactly to the descriptions of Klebs[8] and Friedländer,[8] but many which show this lesion in combination with others.

[1] 'Deutsche Archiv f. klin. Med.,' Bd. lii, s. 339.
[2] Loc. cit.
[3] 'Virchow's Archiv,' Bd. xix, s. 537.
[4] 'Text-book of Pathology,' p. 689.
[5] Loc. cit.
[6] Ziegler u. Nauwerck's 'Beiträge,' Bd. xi, s. 441.
[7] 'Deutsche Archiv f. klin. Med.,' 1880, Bd. xxv, s. 541.
[8] Loc. cit.

The single specimen (Case 10) I unfortunately did not examine by the naked eye.

Microscopically it shows great enlargement of the glomeruli, which all, even in the thinnest sections, appear quite solid. The number of nuclei is enormously increased. Bowman's capsule, including its epithelium, is absolutely normal, and there is no adhesion between it and the glomerulus, since in manipulating sections the latter readily falls out. The other structures are normal.

This condition, discovered by Klebs, was held by him to be characteristic of post-scarlatinal nephritis. His observation was confirmed by Friedländer, but controverted by many other authors. Among those who have worked with an extensive material Cornil and Brault[1] ignore this form altogether, while Hansemann[2] and Wagner[3] expressly state that they have met no such cases.

Lesions sometimes of the glomerular tuft, sometimes of the capsule, are now admitted by most authors to be extremely common in various forms of nephritis, and some even hold them to be invariably present; but in the interpretation of the appearances seen there is great divergence, some holding that in all classes of primary nephritis glomerular changes are the primary, others that the secreting epithelium is the first to suffer. In any particular case where both lesions are found, it seems to me to depend very much on the bias of the observer, which is thought to be the most important. In our ignorance to what extent the slighter variations of structure, whether of epithelium or of glomerulus, prevent their functional activity, it is impossible to compare the gravity of the lesions seen; and except in the matter of quantity, *i. e.* the number of glomeruli or tubules diseased, to state that the one or the other lesion preponderated.

I admit, therefore, that the following cases where glomerular change was found along with more or less extensive affection of the epithelium or stroma are open to a different

[1] Abstracted in Cornil and Ranvier's 'Pathol. Histology,' translated by Miss Hart.

[2] 'Virchow's Archiv,' Bd. cx, s. 52.

[3] Loc. cit.

interpretation from my own, yet I class them with the glomerulo-nephritis, as I believe that that lesion is the most important.

Glomerulo-nephritis with pronounced parenchymatous change.

Case 11.—B. G—, female æt. 9.

March 24th.—Onset of scarlet fever.

29th.—Admitted; temp. 38° C., which persisted until April 10th, when a trace of albumen appeared.

11th.—Vomiting, hæmatemesis.

13th.—More albumen, hæmatemesis continues; very restless; mouth bleeds readily.

14th.—Getting thinner; a profuse erythema.

16th.—Death. Temperature normal since the 11th.

Post-mortem.—Old adhesions of diaphragm to liver and spleen; kidneys large, dark, showing a few white spots in medulla, foci of inflammatory cells. Malpighian bodies easily seen. Microscopically most of the glomeruli impervious, one or two show a slight growth of the capsular epithelium. Tubules almost all dilated, many containing blood or nuclear casts.

Glomerulo-nephritis with pronounced interstitial and parenchymatous change.

Case 12.—Ada B—, æt. 1 year 8 months.

June 2nd.—Onset of scarlet fever.

7th.—Admitted; temp. 38·8°.

9th.—Temperature subnormal; peeling.

10th.—Cloud of albumen. The urine had not been previously tested.

11th.—Vomiting.

12th.—Temp. 39·6°; fair amount of urine.

14th.—More albumen; temperature normal; urine dark; pulse rapid, high tension; vomiting frequent till death on 22nd.

Post-mortem.—Heart weighed 2 oz. Some excess of subarachnoid fluid. Kidneys not enlarged, cortex pale, pyramids dark. Malpighian bodies showing as red clots. Microscopically the majority of glomeruli enlarged and impervious, rich in nuclei. Some show very distended loops; a few have great overgrowth of capsular epithelium and hyaline degeneration of the tuft. Stroma increased and fibrous, especially along the interlobular vessels and around certain glomeruli. Several convoluted tubules have necrosed epithelium, and in some of these a connection with a degenerated glomerulus may be made out. Many tubules are blocked with casts.

The appearances would lead one to suppose that an interstitial nephritis had cleared up, leaving some fibrous tissue behind, and that glomerular and parenchymatous change

had succeeded. But since the duration from the onset of fever was only twenty days the probability of a nephritis having existed prior to the fever must be granted.

In several cases the glomerular change is accompanied by less marked changes in the other tissues of the kidney.

The proliferation of the capsular epithelium as seen in the last case is quite rare in my cases. Even the least developed forms are not frequent. Some observers, *e. g.* Litten[1] and Wagner,[2] regard it as the essential lesion in inflammation of the glomerulus. In view of its commonness in other forms of nephritis, and its rarity among my own cases, all of whom died in the acute stages, I hold that it is probably secondary, and usually follows the loss of function of the glomerulus.

The two types above described are the two extremes between which many other cases may be ranged, as showing both moderate interstitial cell infiltration and obstruction of the glomerular tufts. Yet I find that great confusion of these two still exists. Thus Coats ('Text-book of Pathology,' p. 689) figures a well-marked septic nephritis with periglomerular accumulation of cells as a glomerulo-nephritis; while Ralfe ('Treatise on Diseases of the Kidneys,' p. 158) actually regards acute interstitial and glomerular nephritis as synonymous.

The cause of the impermeability of the glomeruli has been a subject of very lively discussion. Klebs himself regarded it as an overgrowth of connective tissue compressing the capillaries, but the presence of connective tissue in the glomerulus is now generally denied. The modern views are that the new cells are either (1) proliferated epithelium, (2) proliferated endothelium, or (3) leucocytes, or a combination of these. My specimens are, as a rule, not good enough to decide the question; but in some cases I have distinctly made out an excess of epithelial cells lying outside the basement membrane of the capillary, together with an excess of cells which might be either leucocytes or proliferating endothelium within that membrane.

As regards changes not properly belonging to either

[1] 'Charité Annalen,' Bd. iv, s. 179.

[2] Loc. cit.

type, I have seen fibrous thickening of the stroma in one case only besides the case (No. 12) described above. In this one also glomerular and epithelial changes were present, though not so extensive. Death occurred on the thirty-fourth day. In a large number of cases I have seen a few tubes with catarrhal change, the cells being entirely loosened from the basement membrane, and losing most of their protoplasm, while the nucleus remains and stains readily. In one case the majority of tubes were so affected. The glomeruli in this case were chiefly impervious.

Loosening or loss of the cells of the collecting tubes is very frequent, but since it may occur from post-mortem change I do not lay any stress on it.

The changes described and figured by Klein[1] in the arterioles I have not seen. Infarcts were found in a good many of the cases, sometimes solitary, sometimes numerous, and usually associated with infarcts in the spleen or elsewhere. They varied in size, and usually showed an anæmic or necrotic centre with a congested boundary zone. The microscopic emboli found by Klein[1] and Crooke[1] in some cases of interstitial nephritis I have not found.

Bacteria, usually streptococci, have been often found in the scarlatinal kidney by various observers. I have examined a considerable number, by smear specimens, by sections of material hardened in absolute alcohol, and by cultures, with only a few positive results. Most of those I examined were of the septic type, and of these only Case 4 (E. B—, described above) showed a few streptococci in one section. A septic case with nephritis of only moderate severity showed vessels plugged with streptococci. The most extensive blocking of capillaries with streptococci that I have seen was in a specimen prepared by my colleague, Mr. Scholefield, from a glomerular case, and from it I cultivated the *Streptococcus pyogenes*. One would have expected bacteria to be most constantly present in the early septic cases, and less so in the post-scarlatinal; but my own few experiments, together with those mentioned by previous writers, show that the opposite is nearer the truth.

[1] Loc. cit.

Summary.

1. The statistics of the incidence of nephritis among scarlet fever patients under different circumstances are not properly comparable, but so far as any conclusions can be drawn from them they do not show that any diminished incidence results from very strict dietetic treatment during and after the attack of scarlet fever or from prolonged rest in bed. Nor do they show any increase in hospitals during times of pressure.

2. Fatal cases are very rare, forming a little over 1 per cent. at the North-Eastern Hospital, and less at the London Fever Hospital. This refers only to deaths in the acute stage. Death may occur later on from a relapse, but acute symptoms never persist for more than a few weeks.

3. There is no sufficient evidence to show whether hospital routine prevents nephritis. On the other hand, it certainly prevents the occurrence of severe œdema. Even a slight œdema is rare in hospital. The development of œdema has no relation whatever to the diminution or suppression of the urine. Biermer[1] has published a case where complete suppression lasted for five days, and almost complete suppression for five days longer, without any trace of œdema developing.

4. At the onset of an acute attack perspiration is often profuse. If absent, it is always readily elicited by treatment. The dry skin, which has been thought to be characteristic of the disease, does not occur in scarlatinal cases until some days or weeks after the onset, and is by no means constant then. Suppression of the perspiration cannot be the cause of the nephritis.

5. In fatal cases the chief symptoms are continued vomiting, drowsiness (which may later on give place to a restless tremulous stage), anæmia, wasting, and an erythematous rash. Suppression of urine for twenty-four hours or longer sometimes occurs, but is not common.

6. Scarlatinal nephritis is intimately associated with inflammatory affections of other organs. This is true not

[1] Loc. cit.

only of the acute interstitial or septic form, which is never found except in cases with pronounced septicæmia, but also of the post-scarlatinal, which is frequently associated with suppuration elsewhere, and still oftener with non-suppurative inflammations. The clinical evidence in many cases points to the concomitant lesion as the cause of the nephritis, and not *vice versâ*. In other cases they appear to be due to a common cause.

7. In scarlatinal cases the presence of nephritis is an indication of urgency for operations, in contrast to chronic Bright's disease, which contra-indicates operations not in themselves urgent.

8. The kidneys from fatal cases mostly fall readily into one of Friedländer's two forms—the septic or the glomerular; but intermediate forms are not rare. In the latter of the two the solidification of the glomerular tuft may proceed from a combined growth of epithelial cells outside the capillary membrane, and of other cells, whether endothelium or leucocytes, within it. Capsular overgrowth is rare in scarlet fever, and when found only affects a few glomeruli. It is probably secondary.

Glomerular nephritis by itself is not common. It is usually accompanied by other changes, some of which, however, are probably secondary to it.

Kidneys are also found which do not show any obvious cause for the fatal issue of the case.

9. The glomerular form usually shows marked symptoms during life, such as those mentioned above (under Case 5), and also marked changes in the urine. The septic form may be quite latent, but may, on the other hand, show any of the above-mentioned symptoms. The changes in the urine are less marked than in the other form; considerable albuminuria is the exception rather than the rule, and hæmaturia is rare.

DESCRIPTION OF PLATE III,

To illustrate Dr. Meadows Turner's paper on Scarlatinal Nephritis and its Varieties.

Both figures were drawn by the author with a camera lucida under the same magnification, viz. Zeiss Oc. 2, Obj. D.

FIG. 1.—Glomerulo-nephritis. From Case 10. Hardened in Müller's fluid.

a. Glomerulus, much enlarged, showing large increase of nuclei and almost complete obliteration of the capillaries.

b. Bowman's capsule, normal.

c. Convoluted tubes, also normal.

FIG. 2.—Septic or acute interstitial nephritis. From Case III. Hardened in absolute alcohol.

a. Glomerulus showing pervious capillaries. This is not so plain in the drawing as in the original specimen.

b b, *c*, *d d*. Convoluted tubules in different stages of disintegration, the last being mere clumps of cells or even single cells.

The greater part of the drawing is occupied with the largely increased stroma, which is in some parts densely packed with leucocytes; in others it shows a fibrillated coagulum.

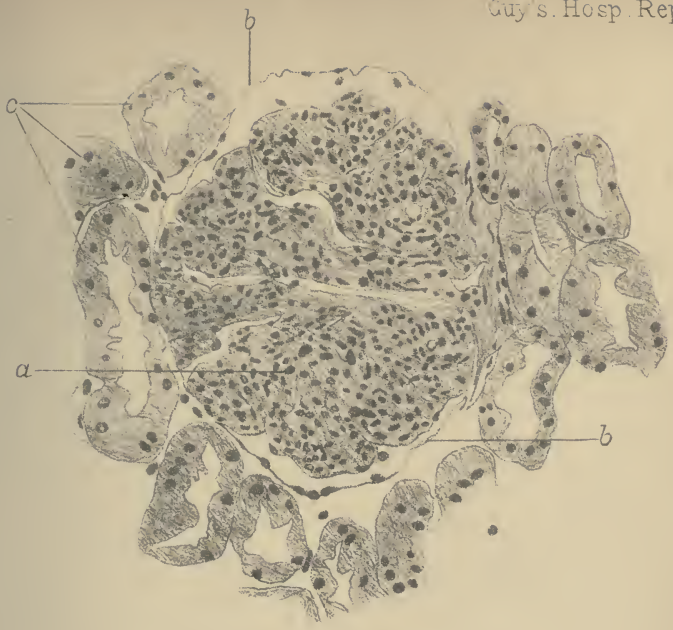


Fig.1. Glomerulo-nephritis.

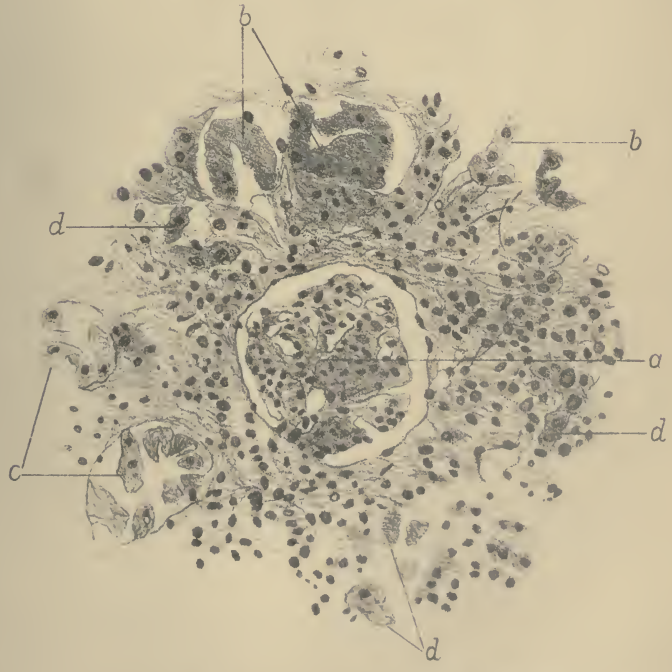


Fig.2. Septic or acute interstitial nephritis.

Danielsson & Co. lith

ON THE

TREATMENT OF CONGENITAL CLEFT PALATE.

By F. NEWLAND PEDLEY.

The possibilities of staphyloraphy as an operation are well known to every surgeon, and daily experience has established the fact that even wide clefts in the hard and soft palate can be bridged over by plastic operations. The three chief physiological functions involved are those of mastication, deglutition, and speech. As regards the effect of staphyloraphy on mastication and deglutition, we can say at once that, provided the cleft can be closed by operation, the patient need not wear an artificial hard palate ("obturator"), and the result is a permanent advantage. But as the faculty of speech is one of the most important natural gifts, and staphyloraphy does not always favour the acquisition of correct articulation, the limitation of cases suitable for operation becomes a matter of great interest.

On the one side we have the strictly surgical view in favour of operation whenever there is a reasonable prospect of closing the cleft, because in successful cases a deformity is permanently removed, and food no longer passes into the nose in the act of deglutition. Even in the instance of a partial failure of union it is considered an advantage

to the patient to have something in the form of a palate, whilst the dental art is invoked to complete the treatment, either by covering over the point of non-union, or by supplying an artificial soft palate called a "velum." On the opposite side the extreme view is expressed by Dr. Norman Kingsley of New York, whose exhaustive treatise on "Oral Deformities" is a standard work of the highest authority. He shows that the chief point to be considered in connection with the operation of staphyloraphy is whether the physiological function of the soft palate can be restored; for unless the patient can, at will, raise the velum palati, so that it acts normally in shutting off nasal resonance, the voice will be marred by the preponderance of nasal sounds, and correct articulation will be impossible. Kingsley thinks that staphyloraphy is rarely justifiable, and he cites one or two cases where it was deemed necessary to partially undo the union resulting from operation in order to facilitate treatment by an artificial velum. Finally, we find him saying of staphyloraphy, "Certain it is that it has been performed many times, when the only apparent object was to gain *éclat* by the skilful use of the knife in a difficult case before an admiring audience, and with no possible hope of even a union or a surgical success."

Kingsley, be it remembered, had devoted many years to

Fig. 1.

Diagram of Stearn's velum, seen in vertical section from behind.
A, A. Lateral leaves of valve, embracing margins of soft palate (C, C), and gliding inwards over the central leaflet (B) during deglutition.

the elaboration of an artificial soft palate or *velum*, which was a modification of one which Dr. Stearn, a native of Massachusetts, America, made for his own palate. But to Snell of London is due the credit of having devised, twenty years previously, a velum that acted on the same principle as Stearn's. The palate worn by Stearn was a marvel of ingenuity and complexity, costing him years of labour for its

completion. It acted on the principle of a triple valve, consisting of a central portion and two lateral leaflets; the latter were grooved along their outer margins to embrace the free edges of the cleft palate, and glided inwards over the central portion when the margins of the cleft approximated in deglutition. Kingsley simplified this velum in a most ingenious way, and made it all in one piece. Too great credit cannot be awarded to him for his work and the admirable book which he published, but his condemnation of staphyloraphy is very uncompromising.

Bearing in mind the conflicting views of numerous writers, I have for some years made a study of the subject, and, as might be expected, one is led to the conclusion that some cases are suitable for operation, whilst others are not, and that various forms of vela will give the best results in certain forms of cleft. For the complete success of staphyloraphy it is essential that the soft palate when united shall be long enough, and sufficiently under the control of its muscles, to perform its physiological function as a valve, enabling a patient at will to prevent the passage of sound from the pharynx into the nose. Class I.—Where the form of the cleft approaches a mere fissure, or in wider clefts associated with fleshy or redundant soft palate, operation may yield a perfect result, and should be performed early in life, so that the subsequent growth of the parts may have a compensatory or remedial influence, and the patient may escape the acquisition of bad habits in voice production. Class II. —Conversely we have the typical case of a broad cleft successfully united after several operations.

The following illustrations are examples of these cases. In Fig. 2 union of the cleft is complete; in Fig. 3 a small opening persists. In both cases the palate is flattened, seamed by cicatrices, the mouth is diminished in size, the soft palate is shortened, rigid, and almost immovable. As far as correct articulation is concerned the operations only increase the difficulties of necessary treatment by artificial means, and I think that staphyloraphy was contra-indicated if the patient could have obtained a velum.

FIG. 2.

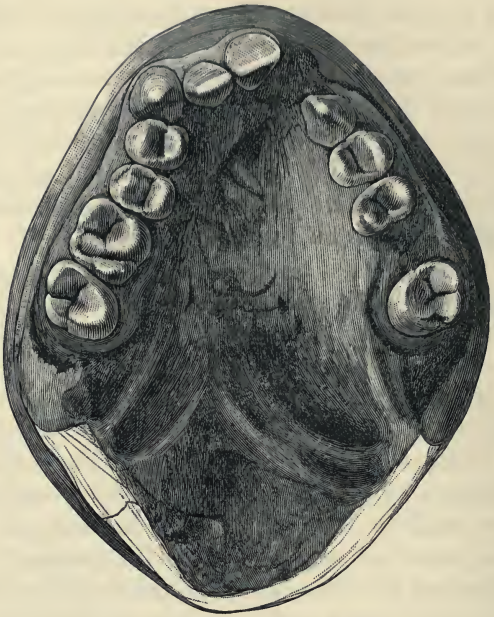
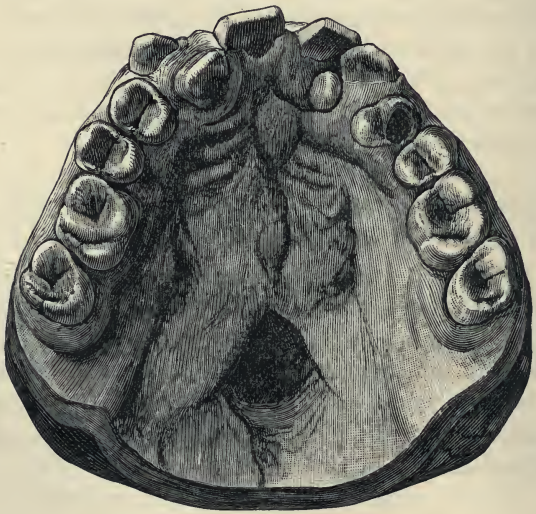


FIG. 3.



Class III comprises the intermediate cases, in which a slight shortening of the soft palate and slight consequent defect of speech would be associated with restoration of the vault of the palate; and here the surgeon's judgment must be guided by collateral circumstances of a very complex nature. Age, sex, position, marriage, means, and individual intelligence must be most carefully considered. Much must depend on whether the patient is in a position to obtain treatment by artificial means; if not, the closure of a cleft by operation is an advantage, even if it be accompanied with slight shortening of the soft palate. If I might venture to quote my own experience in this field, I would say that improvement in the speech, whether as the result of operation or of wearing obturators or vela, mainly depends on the subsequent tuition of the patient, and the results of hospital-practice are vitiated by our inability to carry out the necessary after-treatment. Each patient requires half an hour's instruction twice a week for at least three months to correct the defects in voice production. Those who are fortunately situated can supplement this teaching with lessons in elocution and in singing. They are taught to sing scales and exercises in which they are compelled to open the mouth widely, and to refrain from thrusting the dorsum of the tongue into the palatal cleft. This is a bad habit that all cleft palate cases acquire in their attempt to arrest the predominance of nasal sounds, and they accompany this error with contraction of the compressor nasi muscles. Months or even years may be required to correct these defects of articulation, and great is the disappointment of the surgeon or the dental surgeon when a patient is indifferent to the success of treatment. More than one man is attending periodically at Guy's Hospital, who never shows the least improvement in his speech, and who only values his velum as an aid to deglutition. One of them says that he is tired when he gets home from work, and wants to enjoy himself quietly. Another is an old fellow for whom we have made various forms of obturators and vela, and he is rather pleased to attend the L.D.S. examination as a clinical case. His power of speech improved satisfactorily up to a certain point, but there it stopped. I have been told that he has a son who is quite able to support the

father very comfortably, and it is surmised that he does not wish to speak any better for fear of having to earn his own living.

Of all the cases that I have treated the most successful have been those of young ladies aged about eighteen, who would be described as good-looking. From the time that they know it is possible for them to acquire correct speech they concentrate their whole attention on that object; they requisition in turn all the other members of the family to hear them speak, read, and sing, and even the acquaintance conversing with them unconsciously becomes a useful object lesson.

The mechanical treatment of cleft palate may be commenced at any time after the second molar and the canine teeth are fully erupted. It is an advantage to begin treatment early, because it allows less time for the defects in articulation to become established; on the other hand, the jaw is not fully formed, and patients better appreciate the importance of treatment a few years later, when personal vanity or a desire to engage in some occupation enlists their active co-operation. Numerous are the varieties of obturators and vela which have in turn found enthusiastic admirers, but a careful examination of the different principles, anatomical and mechanical, on which they are constructed will aid us to decide in what cases they should severally be used.

Broadly speaking, a patient will more easily learn to speak with a velum than with an obturator, because a soft flexible artificial palate is a closer approach to the missing normal tissue than any that could be made of hard unyielding material. Experience has established the fact that when speech has been acquired by the use of a velum, an obturator can be substituted for the velum without marked detriment to speech. It is admitted that this result is due to the muscles of the pharynx, the superior constrictor, in especial, having acquired preternatural mobility as the effect of prolonged education.

Luersen's obturator is the best of its kind. It should be hollow and made of hard vulcanite. In shape it has a broad base directed towards the back of the pharynx, which

it almost reaches. The sides of the obturator converge from above downwards, so that the margins of the cleft are kept in apposition with the sides of the obturator, when the edges of the cleft palate are drawn upwards and outwards by the levator palati in deglutition, and in the production of certain sounds. The great advantage of Luersen's vulcanite obturator is that, being made of hard rubber, it does not deteriorate in use, whereas the vela deteriorate very rapidly in some mouths.

Kingsley's velum consists of an artificial soft palate attached to a vulcanite or gold plate. The lateral margins of the velum embrace the edges of the cleft, so that the velum is under the control of the muscles that normally act upon the soft palate.

FIG. 4.

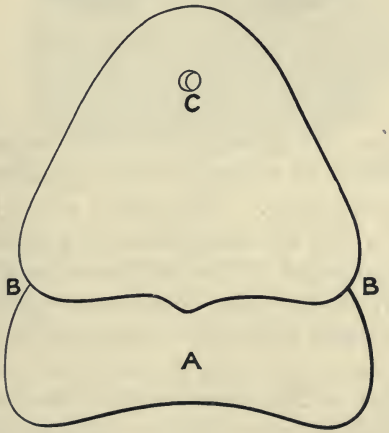
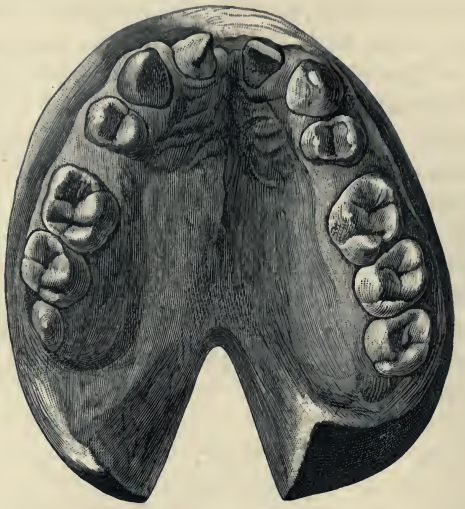


Diagram of Kingsley's velum, palatine surface.

A. Soft palate. B, B. Margins composed of two leaflets of rubber, embracing the margin of the cleft. C. Hole for attachment to metal or vulcanite palate.

The cases most suitable for Kingsley's velum are those where the soft palate is freely movable and controlled by its muscles, but it is just in such cases that staphyloraphy should be considered. Kingsley claims that his velum is best in almost every case ; but when the soft palate is congenitally absent, as in Fig. 5, or is adherent to the side of

Fig. 5.



Congenital deficiency of soft palate.

the pharynx, or has been completely united by operation, I do not think that the advantages of this velum over simpler forms are sufficient to outweigh the far greater difficulty and expense of construction. Sercombe's velum is a thin sheet of soft rubber which can be sewn on to a plate. The centre of this is strengthened by the addition of a smaller piece of thicker rubber, which is also sewn to the plate.

In my out-patient practice I have tried various modifications of this velum, and with great success. Free mobility can be imparted to Sercombe's velum by the adaptation of a light gold spiral spring passing along its upper surface, and attached to the nasal surface of the palate that carries it; the spring keeps the velum in contact with the lingual surface of the patient's soft palate. This velum can be used without a spring when the soft palate is rigid or deficient in quantity. I only use a single thickness of velum rubber, and strengthen the centre by prolonging the posterior margin of the plate into a metal spur to which the velum is sewn.

Metal is better than vulcanite for this purpose, because it can be bent upwards or downwards in adjustment.

Fig. 6.

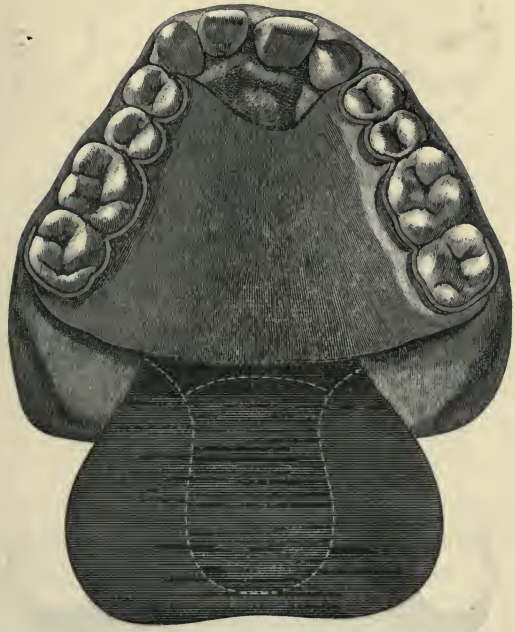


Diagram of Sercombe's velum palati surface.

In hospital practice Sercombe's velum has the immense advantage that patients can themselves cut out and sew fresh vela on to the plate. This is a consideration amongst those who wish to be supplied with artificial palates for the rest of their lives.

Parkinson's artificial palate should be mentioned. It consists of the usual plate covering the hard palate, to which an artificial velum palati made of hard vulcanite is attached by means of a hinge and spring. The hard rubber does not deteriorate in the mouth, but is not so adaptable to surrounding parts as a soft velum would be.

There are other contrivances, solid, hollow, or even inflatable, made of various materials, and attached by springs to the nasal or lingual surface of the palate.

I purposely omit any reference to the processes by which these appliances are made, or to the difficulties that will arise in diverse cases; enough has been written to show

Fig. 7.

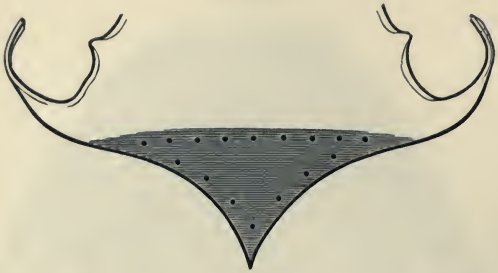


Diagram of plate prolonged into metal spur (shaded).

Fig. 8.

Diagram of Parkinson's artificial palate, nasal surface, attached by means of a spring and hinge.

some of the general principles on which congenital clefts may be treated, and the relative special advantages of certain artificial palates.

ON THE

ADMINISTRATION OF GAS AND ETHER.

By J. FREDERICK W. SILK, M.D.

Of modern methods of inducing surgical anæsthesia probably the most complicated, and certainly the most difficult to master, is that known as the "gas and ether" method, in which advantage is taken of the properties peculiar to nitrous oxide and ether respectively, in order to produce a result which, in some particulars at any rate, shall be superior to the results obtained when either of these substances is used separately.

No method of administration is so apt to be rejected after the first few trials on account of its apparent uncertainty, and no method requires more constant practice in order to maintain and develop the skill already obtained. For these reasons it might almost be held that mere verbal descriptions are of little value, and such descriptions must certainly be considered as of quite secondary importance to practical demonstrations and experience; but, on the other hand, it may not be altogether out of place to attempt to give some account of the general principles involved, to consider exactly what are the objects of the combination and how they are to be attained, and what, if any, are the limitations or objections to its use.

The special peculiarities of the anæsthesia induced by nitrous oxide alone may, for the present purpose, be taken

to be as follows:—The gas itself is unirritating, and has a rather pleasant taste and smell, not only, therefore, do patients seldom object to inhale it, but it does not cause any respiratory spasm; its action is rapid, owing to the transient character of the stage of excitement and struggling; it is usual to describe it as having but a feeble anæsthetising power, so that to produce its full effects it must be given in such a way that all air is excluded,[1] with the result that towards the end of the administration symptoms of asphyxia or oxygen starvation are developed; its administration is not associated with or followed by any marked degree of depression, so that recovery is rapid, complete, and not accompanied by nausea or sickness. Practically, therefore, nitrous oxide is not suitable for the maintenance of anæsthesia for any length of time, and its use is limited to those operations which can be completed in the short period which the primary induction affords, or, in other words, to a single application of the face-piece.

The peculiarities of ether anæsthesia are:—The vapour is very repugnant to most people, and almost always irritating, tending to cause respiratory spasm; the stage of excitement is very pronounced; it is a much more powerful anæsthetic than nitrous oxide, and can therefore be administered with sufficient air to prevent the development of oxygen starvation symptoms; as compared with nitrous oxide recovery is slow, and usually associated with sickness and other unpleasant after-effects.

The late Mr. Clover was the first, or one of the first, to recognise the importance of combining these two substances, and the inhaler which bears his name was originally devised by him for this purpose. To this combination the somewhat generic term "gas and ether method" was applied; and, broadly speaking, the object was to retain the rapidity, pleasantness, and freedom from irritation and excitement of the nitrous oxide anæsthesia, and at the same time take advantage of the greater strength of the ether. But it is

[1] The advantages of the plan of introducing minute and measured quantities of oxygen or air, as advocated by Dr. Hewitt and others, can hardly as yet be said to be sufficiently well established to come within the range of practical anæsthetics.

obvious that as there are two substances concerned, the matter may more properly be considered from the point of view of either of them. We may, for instance, look upon the ether as merely an adjunct to the nitrous oxide, or we may consider the nitrous oxide as simply an accessory to the ether; and as a matter of fact I believe myself that the mode of action of the combination is not precisely similar under these two circumstances, and therefore that the principles involved, as well as the actual details of administration, vary slightly according to whichever of these objects we have in view.

Looking at the matter first of all from the point of view of the nitrous oxide, the sole object which we have in view in adding the ether vapour is to eke out the anæsthetising strength of the gas, and so obtain a little longer time for the work of the operator. This method may be called the method of etherising the nitrous oxide (for the sake of brevity we may represent it by the letters Ne), or it is sometimes spoken of as "gas with a whiff of ether." In this plan not only is the main anæsthetising agent still the nitrous oxide, and not only do we take advantage of the rapidity, pleasantness, and freedom from irritation and excitement of the nitrous oxide anæsthesia, but in increasing the anæsthetising strength by the addition of ether we endeavour to avoid those disagreeable after-effects which frequently follow the use of ether alone, and to arrange matters so that the recovery shall be almost if not quite as rapid, complete, and free from nausea, &c., as when gas alone is employed. To obtain these satisfactory results it is, I believe, very essential that the nitrous oxide anæsthesia should be pretty far advanced before any attempt is made to add the ether vapour, and that when the latter is introduced it should be introduced in considerable strength, and the proportion rapidly increased.

When nitrous oxide is given by itself the time taken to induce narcosis is approximately seventy seconds, and the resulting anæsthesia is said to average about thirty-six seconds. When ether is added in the manner suggested above, induction is often slightly prolonged (possibly on account of air-leakage due to the use of a more complicated apparatus),

and the available anæsthesia is increased by about half as much again, *i. e.* to about fifty-four seconds. One of the peculiarities of the anæsthesia induced in this way is, that even after the patient becomes conscious, he will often submit quite quietly to manipulations of the wound, particularly if about the mouth, which would be violently resisted when recovering from the lighter anæsthesia of nitrous oxide; and in the same way the after-soreness of the operation is not developed so early or so severely. Although the effects of etherising the nitrous oxide in this way are sufficiently decided, I cannot help thinking that the action of the ether is partly a local one, and only partly due to the presence of the ether in the blood. The time during which the ether is inhaled is so short, especially in proportion to the effects produced, as to render it very improbable that any large amount of ether can have been absorbed. Beyond perhaps a slight temporary headache or a transient feeling of nausea, there are or should be no disagreeable after-effects; directly, however, an attempt is made to obtain a more permanent anæsthesia these after-effects are apt to develop.

It may perhaps be objected, that for the use of etherised nitrous oxide the field must be very limited. To some extent, no doubt, this objection holds good; but I believe, nevertheless, that it is this method which, voluntarily or involuntarily, is employed in dental work when only a slight increase in the duration of the anæsthesia is required. In surgical work I have found the plan of value in such cases as scraping the naso-pharynx for adenoid growths, removal of small spurs of bone from the nasal septum, perforating the antrum of Highmore, &c., when the local effect is certainly of value, and in such very short cases as moving stiff joints, opening small abscesses, &c., when the general effects are relied upon. Apart, too, from the prolongation of the anæsthesia, I have found the stimulating effects of the ether to be of advantage to those patients whose feeble circulation not only renders them liable to syncope, but also tends to shorten the duration of the anæsthesia obtainable from the use of nitrous oxide alone.

Looking now at the matter from the point of view of the

ether, the main object which we have in view in using nitrous oxide in conjunction with ether is to avoid some, if not all the unpleasantness which characterises the induction of anæsthesia by ether vapour alone, without sacrificing the continuity of the narcosis or limiting its duration ; this method may be termed the method of nitrogenising the ether (for the sake of brevity it may be represented by the letters En). In this plan the anæsthetising agent upon which we really depend is the ether ; the nitrous oxide is merely used as a vehicle for conveying a sufficient strength of the pungent ether vapour to the lungs. We want to induce the anæsthesia rapidly and without irritation or excitement, but we do not attempt by this plan to obviate or overcome the after-effects of the ether anæsthesia.

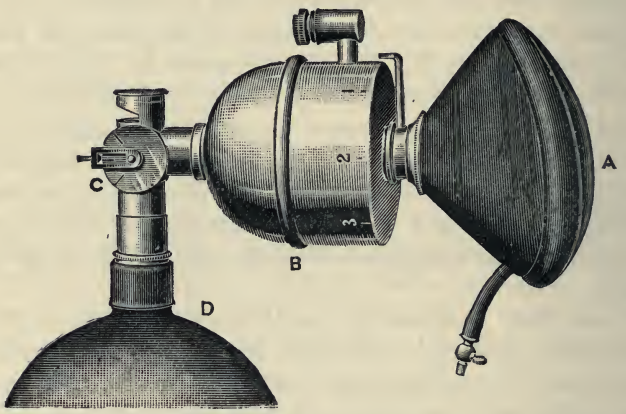
To obtain these results it is, I believe, most important that the ether vapour should be added to the nitrous oxide so gradually that the patient shall not be conscious of the addition, nor give any involuntary signs, such as cough, respiratory spasm, &c., of the presence of the irritating vapour. Another important principle is, I think, that we should so time the increment of the ether vapour as to ensure that by the time the asphyxial symptoms (subsultus, stertor, &c.) of the nitrous oxide have developed, the patient should be breathing a sufficient percentage of ether vapour to maintain the anæsthesia without any break in its continuity.

When ether is given by itself the time of induction is between four and eight minutes ; when nitrous oxide is used as a vehicle it is seldom that more than two minutes are required : add to this the absence of excitement and respiratory irritation, and above all the far greater comfort to the patient, and I think that the advantages of this method will be at once apparent, and, in fact, that by the use of nitrous oxide we can overcome not a few of the objections which have been raised to the use of ether alone.

In addition to these two classes of cases there are, of course, many which occupy an intermediate position, and concerning which it would be difficult to say whether the ether or the nitrous oxide was the predominant anæsthetis-

ing agent, but these intermediate cases are not, I think, so numerous as is usually supposed.

Whether we wish to etherise the nitrous oxide, or to nitrogenise the ether, it is of advantage to accustom one's self to use the same form of apparatus, so that, should necessity arise, we can the more readily prolong the narcosis by converting what in one case is practically a nitrous oxide anæsthesia into an ether one. The apparatus which I prefer myself is that known as Clover's large gas and ether apparatus, made by Messrs. Meyer and Meltzer; this instrument is an excellent one, but somewhat complicated, and apt to get out of order unless kept in constant use. For practical purposes I am in the habit of recommending Clover's small or portable inhaler, so modified as to permit of its being mounted between the face-piece and the three-way tube of an ordinary nitrous oxide apparatus (see figure); the modifica-



A. Face-piece. B. Ether Chamber. C. Threeway tube and valves. D. Reservoir bag.

tion consists essentially in a slight prolongation of the central tube, so as to avoid the difficulty of the whistle-ended tube usually supplied. In using this apparatus the chamber is first charged with ether, the index turned carefully to the 0, the reservoir bag filled with nitrous oxide, and the face-piece carefully adjusted to the irregularities of the face; the nitrous oxide is then turned on, and the inhalation proceeds as in an ordinary gas case. If we simply

intend to etherise the nitrous oxide, the inhalation is permitted to continue for something like ten or twelve full respirations,—that is, until the increasing lividity and harsh but regular breathing indicate that the patient is passing under the influence of the gas. Arrived at this stage, the ether chamber is rapidly turned, so that in the course of the next four or five respirations the index points successively to the 1, 2, or even 3, a pause being made at each additional onward movement, so as to avoid respiratory spasm. By this time there will usually be some symptoms of oxygen starvation (twitching and irregular stertor), and the face-piece must be removed and the operation can be commenced.

If we intend to nitrogenise the ether, the first movement of the ether chamber is made after about five or six full respirations have taken place, when we may fairly assume that even if the patient is not absolutely unconscious, at any rate the sensibility of his mucous membranes has diminished. This first movement should be very slight,—not more, perhaps, than one eighth of an inch; the next movement may be a quarter of an inch, the next half an inch, the next three quarters of an inch, and so on by progressive increase, in every case of course a pause being made on each addition to accustom the patient to the increased strength of the vapour. Proceeding thus cautiously, we ought to be able to work the index up to about the 3 without having had any indication of oxygen starvation; keeping the index at the 3, the threeway stopcock may now be turned to permit to-and-fro respirations into the bag, and in any event, on the first indications of twitching or irregular stertor a little air may be admitted under the face-piece, and the bag emptied of gas; or the same purpose will be served by changing the somewhat cumbersome gas-bag for a smaller and lighter one. In a typical case the patient should now be well under control; he is breathing a strength of ether vapour more than sufficient to maintain the anæsthesia, and the lividity, twitching, spasm, &c., symptomatic of the latter stages of nitrous oxide anæsthesia, *i. e.* oxygen starvation, will rapidly subside under the influence of a breath or two of air.

I am bound to admit that this description of the details

of the administration reads much simpler than it will be found to be in actual practice, and that, especially in the hands of the novice, these typical cases are rarer than one could wish; and this brings me to the consideration of the most common difficulties and troubles connected with this particular method of administration. It is obvious that where so much depends upon the skill and judgment, and therefore upon the experience of the individual administrator, it is impossible even to refer to all the difficulties that may arise; I can only allude to those which have appealed most strongly to myself.

In etherising the nitrous oxide the ether may be admitted too late or too slowly, with the result that its full effects are not obtained, and the face-piece has to be removed before a sufficient strength of vapour has been administered. For this, of course, there is no remedy as far as the individual case is concerned, but a few cases of this sort serve to emphasise the fact that each case must be judged upon its own merits, and that neither this nor any other method of administration can be conducted by rule of thumb.

On the other hand, the ether may be added too quickly, in which case of course an acute condition of respiratory spasm may result, or the development of oxygen starvation symptoms may be somewhat anticipated. In these latter cases I do not think that it is absolutely necessary to remove the face-piece at once and entirely. If the spasm is due simply and solely to too great a strength of the vapour, this spasm will subside when the tissues become accustomed to the presence of the ether; but if not, or if there is any doubt in the matter, a little air admitted by lifting one edge of the face-piece will usually restore the equilibrium. In a third class of cases, occurring mostly in children, I have noticed a tendency to over-narcotism without the development of respiratory spasm, twitching, or other pronounced symptoms. The lividity is maintained, the respirations become shallow and feeble, there is complete muscular relaxation and conjunctival insensibility with dilating pupils. These cases are, I fancy, dangerous ones, and point to the necessity of carefully watching the patient during the whole time of the administration; and further

they indicate, to my mind, the fallacy of relying upon any one sign, such as the subsultus, rather than upon the general condition. As a rule, to which I have myself seen no exceptions, recovery after removal of the face-piece is rapid and complete, but I can well imagine that carelessness might result in disaster.

In nitrogenising the ether the difficulties vary in degree rather than in kind from those already referred to. If the ether is turned on too late, muscular spasm, &c., may occur before a sufficient strength of vapour has passed into the lungs; if, on the other hand, the ether is added too soon, the patient will more or less consciously resent the addition. In either event air must be admitted, when the benefits of this method are wholly or partially counteracted. I think I have noticed, too, that the semi-unconscious struggling which results from these errors is often very violent,—more so, perhaps, than when ether alone is employed. Although in my description I have referred to the possibility of abolishing all excitement or struggling, and although I believe that this is certainly possible in a large number of cases, yet in no inconsiderable number of patients one may detect some slight signs of this stage after the gas has been removed. Under the circumstances this is not very surprising, and the probable explanation appears to be that the ether, although present in a high percentage in the lungs, has not had time to exert its full influence upon the cerebral centres; and when the restraining influence of the nitrous oxide is withdrawn, the effects of the ether, though perhaps modified, tend to follow the ordinary course. From what I have said it will, I hope, be apparent that in the combination of nitrous oxide and ether we have a most valuable addition to our list of available methods of producing anæsthesia, and I would conclude by adding that this combination is possible in all cases in which either nitrous oxide or ether by itself is considered suitable.

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DESCRIPTION OF PLATE IV,

Illustrating Dr. Pye-Smith's paper on Bilateral Paralysis of the Facial and Auditory Nerves.

FIG. 1.—Drawing of the patient A. C— when at rest.

FIG. 2.—Photograph of the same patient with attention aroused, the eyes widely open, and the head thrust forward. The node on the left frontal bone is also seen.

FIG. 3.—Photograph of the same patient with the eyes closed as much as possible, and with the lower lip pushed up.

A CASE

OF

BILATERAL PARALYSIS OF THE FACIAL AND AUDITORY NERVES.

By P. H. PYE-SMITH, M.D., F.R.S.

A. C—, æt. 24, was admitted into Philip Ward, No. 30, on the last day of 1894. He was born at Glasgow, and lived there till he was sixteen. He then came to London, working as an unskilled labourer until he was twenty-one, when he enlisted in the army and was immediately sent to India.

Soon after his arrival at Calcutta he contracted a venereal sore, which was followed by a rash on the trunk and arms. This the patient "believed to be prickly heat, for it was treated with calomel." His general health continued good, notwithstanding several attacks of malarial fever, until nine months ago, when he was ill for three months (April, May, and June, 1894) with what was called "enteric fever, followed by slow continuous fever." During this time he was scarcely able to eat anything, and his weight dropped from 10 stone 12 lbs. to 6 stone 8 lbs. Early in his illness he noticed a swelling on each side of the scalp, which slowly spread forwards; and about the same time he became deaf

and lost power over the muscles of his face. After three months the fever left him (July last), but being unfit for service he was invalided and sent home to Netley Hospital, where he arrived in November, 1894. He stayed there a month, improved in strength, and lost a giddiness which before had prevented his walking; but the deafness and facial paralysis remained unaltered.

He came to Guy's Hospital, as an out-patient at the end of December, and was seen by Dr. Pitt, who, recognising the unusual character of the case, sent him into my ward.

On admission the temperature was 98·1° F., the pulse 84, and respiration 15. The heart, lungs and abdominal organs, the skin and the urine showed no signs of disease. There was complete facial paralysis of both sides and absolute deafness.

The following account is drawn up from my own notes, and from an excellent report by my ward clerk, Mr. Robert Howard.

The patient is a young man of average height, 5 feet 7½ inches, well built, but still somewhat thin (133 lbs., or 9½ stone), his complexion remarkably dark, his lips thick, the bridge of his nose depressed, and his hair black and bristly with a few white hairs scattered among it. My first impression on seeing him was that he was not of pure European blood, but apparently both his parents were born in Scotland.

When he is at rest, the paralysis would almost escape notice, for the only sign is drooping of the lower lip, so as to show more of the prolabium than usual, and to expose the lower incisors and part of the gums. He looks like a patient who is suffering from salivation, and his sallow, swarthy complexion, motionless features, and taciturnity give him a peculiarly stolid and melancholy aspect (Fig. 1). The eyes are very dark, and the conjunctivæ dingy. The movements of the eyeball and of the levator palpebræ are perfect. The fundus is normal, and the sight unimpaired. When bidden, by gesture or by writing on a slate, to close his eyes, the upper lid falls a little lower, but the palpebral fissure remains about a quarter of an inch broad (*see* Fig. 3). At the same time the well-known correlated movement of

the superior rectus and inferior oblique muscle rolls the eyeball upwards, so that the sclerotic alone is visible.[1] The forehead is perfectly smooth, and cannot be wrinkled by the occipito-frontalis or the corrugator supercilii muscles. The skin of the face feels soft and inelastic. The patient has perfect control over the muscles which move the lower jaw, and the tongue, including its tip, can be freely moved in any direction; but there is paralysis of the buccinator, shown by his inability to whistle or to blow out a match, and by food collecting between the jaws and the cheeks. The pupil acts perfectly to accommodation and to light.

There is no paralysis of the soft palate or uvula.[2] The mouth is dry, the saliva scanty and viscid, neutral or slightly acid. The patient is aware of this, and complains of thirst.

Deafness is complete; the patient cannot hear a watch held in contact with the skull any more than when held close to the external meatus. He says that he often hears a ringing or tinkling sound like that of a bell heard at a distance.

He seldom speaks, but the laryngeal voice is normal. The articulation is perfect for the vowels and for all the consonants except the labials—P, B, F, V, and M. These he is unable to pronounce, and either omits their proper sounds or replaces them by an aspirate or a guttural aspirate. By pressing up the under lip with the finger he is able to remedy this defect of speech, and the labial sounds are then perfectly produced. So small is the weight of the lower lip, that the mere adhesion of its moist surface to that of its fellow is enough to keep it in position for an appreciable time after the support of the finger is withdrawn,

[1] As Todd observed, this does not accompany gentle closure of the eyes as in sleep, but only forcible contraction of the orbicularis.

[2] It is remarkable how this symptom, fully described by Romberg, has been regarded by subsequent good observers. It is stated to be constantly, or at least regularly, present by the late Dr. Sanders of Edinburgh, by Davaine, Erb, and Eichhorst; while Dr. Todd thought it was only occasionally present, and its occurrence is doubted or denied by Wilks, Ross, Bastian, and Gowers. I was taught to look for it as an ordinary symptom of Bell's palsy, but have never satisfied myself of its presence. The uvula is often a little curved when drawn up in perfectly healthy persons.

long enough for one of the labial consonants to be pronounced.

The spleen can be felt below the costal margin; it does not come forward in the more frequent way, but extends directly downwards toward the iliac crest. The dulness measures vertically $4\frac{3}{4}$ inches from the ninth costal interspace to an inch above the ilium. This enlargement of the spleen was no doubt due to malaria, and threw no light on the paralysis.

When admitted, the case was described as one of "deafness with facial paralysis and ataxia." Ataxia, or want of co-ordination of the legs in walking, was certainly present, but it was not the locomotor ataxia of Duchenne. It depended, not on any lesion of the cord or of the sensory nerves, but on auditory vertigo.

The patient's arms and legs, though they have not regained their former bulk, are still muscular, and he has complete control over his movements as he lies in bed. He can judge of weight and direct his feet or fingers with perfect accuracy. He walks strongly, but with a peculiar gait; after making a few steps he will roll to one side, and in recovering himself will cross one leg over the other, or separate his legs widely and roll in the opposite direction, according as the loss of balance occurs later or earlier in making a step. The lurch is more often to the left than to the right. When it happens, the patient seeks to redress his balance by putting out his hand. The movements of the feet and the character of the walk are quite unlike those seen in tabes; they approach nearer to the reeling gait seen in many cases of cerebellar disease. The patient's walk is less irregular and blundering than that of drunkenness, and rather resembles that of a landsman on board ship when the vessel is rolling. When the patient is blindfolded and stands with his feet together, he begins, often almost immediately, and always in less than a minute, to sway from side to side, and would fall if he did not extend his basis of support by separating his feet. When walking blindfold his gait is more unsteady than with his eyes open, but its reeling character is the same. He says himself that he cannot walk steadily because he feels giddy, his head swims,

and he feels as if he were drunk. As with other kinds of loss of control over muscles, it is worse when he goes into a dark room or walks suddenly out of the shade into bright gaslight.

The skin of the face and body is normally sensitive to touch, to heat and cold, and to pain, with the exception of hyperalgesia to the electric current on the face. The superficial reflexes are normal except the cremasteric, which is somewhat deficient. The knee-jerks are brisk, but clonus is absent. The portio dura of each side does not respond either to the faradic or to the interrupted galvanic current. The muscles it supplies do not contract in response to faradism, but contract slowly to galvanic stimulation. For the orbicularis oris and other labial muscles KCC = ACC. For the orbicularis palpebrarum KCC < ACC. On the right side the kathodal closing contraction was obtained with three cells, the anodal closing contraction with five. On the left both were obtained with three cells. No contraction of the occipito-frontalis could be obtained with five cells, though the current thus obtained was painful. Thus reaction of degeneration was incomplete (the intermediate degree, as Erb calls it), but the process was already considerably advanced; and this agreed with the date to which the patient assigned the paralysis.

In addition to the complete deafness and motor paralysis, it was interesting to note that the sense of smell was considerably impaired, possibly from the patient being unable to sniff. He could not detect the scent of camphor or lavender water.

The sense of taste was also defective. He could recognise salt solution and vinegar, but strong syrup tasted like water, and quinine in solution was once said to taste sweet, and twice to taste salt. This symptom, like the foregoing, has been often observed, and ascribed to paralysis of the lingual branch of the facial nerve.

Although vision was normal the patient complained of his eyes watering, particularly if he held them down. This also is a symptom long recognised, and correctly described by Duchenne as due to the puncta lacrymalia being everted from paralysis of the tensor tarsi. There was no ulceration of the

cornea. He thought that though he could see near objects plainly, those at a distance were less distinct since his illness.

Apart from the difficulty of communication and the immobility of his features, the patient seemed to be dull, and was often confused in his answers. He gave no trouble in the ward, and there was no reason to suppose that his intelligence or temper had been altered by his illness.

The case was clearly one of paralysis of the portio dura and auditory nerves on both sides, with a kind of vertigo and "auditory" ataxia, which may be reasonably attributed to loss of the afferent impressions from the semicircular canals.

The exact seat of the lesion was less clear. It was not in the terminal distribution of each facial nerve, for then we should have to suppose that a degenerative process had advanced up to the motor nucleus of the portio dura, and had thence travelled to the sensory nucleus of the portio mollis and affected that nerve also. There was no evidence of disease of the petrosal bone on either side, and the membrana tympani was intact in both ears.

I was at first disposed to assume the existence of a central lesion affecting the nuclei of both divisions of Willis's seventh pair of cranial nerves; but a tumour or other gross lesion was excluded by the absence of severe symptoms, and a disseminated sclerosis could scarcely have picked out the several nuclei involved without also affecting the adjacent nuclei in the pons and bulb, particularly those of the sixth and of the motor branch of the fifth nerve. On the whole it seemed most probable that the seat of the lesion was in the internal auditory meatus on each side.

The nature of the lesion was more readily ascertained than its seat. The lymph-glands in the groin were slightly enlarged, hard, and painless. There was a similar one in the occipital region. There was a pigmented patch on the abdomen and a node on the orbital process of the left frontal bone. These facts needed no confirmation from the history of venereal disease given by the patient. In the absence of any traumatic or other infective cause it appeared certain that the paralysis was syphilitic in origin.

The presence of a gumma in the pons, or of basal meningitis, seemed to be improbable from the escape of the ocular and other cranial nerves. The lesion was pretty certainly situated between the superficial origin of the seventh and eighth nerves (of Sömmering) and their separation at the distal end of the internal meatus. In this narrow space a symmetrical inflammation of the fibrous sheaths of both nerves was possible, and syphilis supplied an adequate cause.

Accordingly the patient was put upon a course of iodide of potassium, and after a fortnight he felt decidedly better. He said his head was clearer; he was less giddy, and could walk more steadily. The dose of iodide had been raised from 10 to 15, and then to 20 grains three times a day. During the second half of January it was increased to 120 grains every day. This did not produce a rash or salivation but there was some injection of the conjunctiva.[1]

Notwithstanding a slight supposed improvement in the patient's power of closing his eyes, the paralytic symptoms remained practically unaffected under these full doses of iodide; and, what was more surprising, the node on the left eyebrow increased, and a fresh one appeared on the left parietal bone, while tenderness of the scalp on percussion was not at all diminished. On the 31st of January the nodes, now three in number, were thought to be rather smaller; they were still tender, and some coryza was present. The prescription was then altered to the combination of the three iodides recommended by Mr. Hutchinson; 15 grains of each were taken three times a day, and mercurial liniment was rubbed into the neck and scalp. On the 2nd of February the temperature, which had been normal or subnormal until four days before, had risen to 100·8° F. The node on the forehead was larger, softer, and more tender.

On the same day my colleague, Mr. Purves, kindly examined the patient's hearing. He confirmed the absence of any sign of disease of the tympanum. The deafness, he

[1] Iodides were readily demonstrated in the urine by adding hydrochloric acid, and then warming with a few drops of nitric acid and adding starch; the urine became dark brown from the reduction of iodine, and gave a blue colour with starch solution.

said, was nervous; but he thought it possible that it was unconnected with the facial paralysis, and due to excessive doses of quinine when the patient was in India.

Early in February the patient had gained in weight and was feeling stronger. The dryness of his mouth from paralysis of the chorda tympani continued unchanged. "The patient, though readily obeying directions which are conveyed by writing or by gesture, has always been averse from speaking. On making him read, the vowel sounds are harsh and somewhat nasal, probably due in part to his deafness. The labial sounds—P, B, F, V, and M—are, as before noticed, unpronounceable, unless he keeps his lower lip up with his finger. Some acneiform pimples have appeared on the face."

On the 9th of February fresh indurated occipital glands were to be felt, and no improvement had taken place; the exhibition of iodides was therefore discontinued, and the solution of perchloride of mercury in doses of fʒiss was given three times a day. Under this treatment the coryza and iodide rash disappeared, as did the iodide reaction in the urine; but no change in the paralysis occurred, although the dose was increased to ʒij. The large node on the left eyebrow, which had gradually increased and softened, now, however, began to subside, and became decidedly less tender.

On the 11th of March there were two small hard occipital glands, a small node to the left of the occipital protuberance, another on the right side of the same bone, a larger one above the left ear, and the original one above the left eyebrow. A small superficial ulcer was noticed on the right cornea, and the eye was covered with a pad to prevent this mischief spreading. At the end of the month the course of mercury was stopped, and iodide of potassium again ordered in compound decoction of sarsaparilla.

Soon after this the patient left the hospital without leave, and for no ascertained reason. It must be admitted that the treatment adopted had completely failed in its principal object.

Nevertheless there can, I think, be no doubt that the diagnosis was right as regards the nature of the disease,

though its exact locality may leave room for difference of opinion.

Happening to mention this unusual case to Sir William Roberts, he was good enough to refer me to two very similar cases which were reported by him in the 'British Medical Journal' for October 4th, 1862 (vol. ii, p. 355).

The following is an abridged account of the two cases:

(1) A. M—, æt. 47. "A tall, spare man, of vigorous frame. His wife supplied me with the following particulars of his previous history. He has been a healthy man of somewhat intemperate habits. About a twelvemonth ago his health gave way, and he was reduced to a state of considerable weakness. One day about this time he was seized with a sort of fit, which only lasted a few minutes. Whether it was syncopal or apoplectic is impossible to make out; it left, however, no consequences behind.

"A fortnight after this fit the patient, on awaking in the morning, discovered that he was not able to move the right side of his face nor to close his right eye. Eight days later, suddenly in the evening, the left side of the face became paralysed, and the features, previously distorted and drawn to the left, resumed their symmetry. All motion of the face had now ceased. An important circumstance occurred between the two seizures. Four days before the second attack the patient became conscious that he was totally deaf on both sides. He is positive that the deafness supervened in the interval between the two seizures of facial paralysis. It appears, on inquiry, that he was always hard of hearing on the right side, so that it is uncertain whether total deafness of that side preceded or accompanied the first paralytic seizure. From the date of these two attacks up to the present time there has existed without change total deafness of both ears, and total paralysis of the muscles of facial expression. No other part of the body shared in the palsy, and feeling is perfect in the integuments of the face. The general health has greatly improved since the date of the attacks, and for many months now the patient has been free from bodily illness. He complains, however, of some cephalalgia and of a sense

of weight and tightness over the forehead. The skin of the forehead and of the nose have a thickened congested appearance, and seem to have suffered repeated attacks of fugitive erysipelas. There has not been any running from either ear at any time."

(2)[1] T. F—, is a labourer fifty years of age. Twelve years ago he contracted a chancre, which went on to phagedænic ulceration, and has left to this day a deep and extensive scar on the glans penis. Six months after this event he suddenly lost the power of the right side of the face, and six weeks later the left side of the face became similarly affected. Two days after this latter occurrence he became stone deaf. The deafness came on suddenly; to use his own expression, "in one hour."

He cannot tell whether he was deaf of the right ear during the six weeks that intervened between the seizure of the two sides of the face.

Dr. William Roberts, in commenting on these two cases, remarked upon the immobility of the countenance, which forms a mask without change of expression, on the gravity and fixed solemnity, the mournful immobilities of the expression thus given. He noticed that tears easily trickled over the cheeks from paralysis of the orbicularis and tensor tarsi, a symptom only slightly marked in my own case; also the imperfect closure of the eyes during sleep when threatened by an approaching finger, the complete escape of the tongue and almost complete escape of the palate, the salivation, and the defects of articulation. He also states that one of the two patients (T. F—, the elder one) found difficulty in swallowing when first paralysed, and was still sometimes embarrassed by small fragments of dry food. This last symptom was not present in my patient—at least when he first came under observation.

Dr. Roberts discussed the seat of the lesion, and decided against its being either central or peripheral (in the restricted sense of the word,—physiologically speaking, in the motor end-plates), and against its being in the course of the

[1] This second case was exhibited by Mr. Thomas Windsor to a meeting of the Manchester Medical Society in October, 1861, and given to Dr. Roberts for publication.

DESCRIPTION OF PLATE V,

Illustrating Dr. Pye-Smith's paper on Bilateral Paralysis of the Facial and Auditory Nerves.

Photograph of Mr. Windsor's patient, T. F—, reported in 1852. From a negative kindly given the writer by Sir William Roberts.

portio dura through the Fallopian canal. In fact, the same reasoning by exclusion which I afterwards followed led Dr. Roberts thirty years ago to fix the seat of the lesion with the same confidence in the internal auditory meatus.

In his commentary on the two cases reported Dr. Roberts next considered the probability of some general cause affecting both nerves, and comes to the conclusion that in all likelihood syphilis was the cause in question. It was certainly present in the latter of his cases, and probably in the former.

In my own case we had the best possible evidence, actual as well as historical, for the same conclusion.

Of other facial conditions which might lead to paralysis of the seventh and eighth pair of nerves on both sides, improperly, as I think, called "diatheses" (*cf.* vol. xix of these Reports for 1874, p. 319), some are toxic like plumbism and mercurial or arsenical poisoning, which are practically here excluded. Chronic alcoholic poisoning leads to peripheral neuritis with paresis on both sides, but neither it nor plumbism affects the cranial nerves. There was no gout present in these cases, and though it also may cause symmetrical peripheral neuritis, its action also does not extend beyond the limbs. "Rheumatism" in its proper sense of rheumatic fever does not affect fibrous tissues in general, and certainly not the sheaths of nerves. Tuberculosis of the pia mater over the roots of the nerves in question would have produced more extensive lesions and more general disturbance: a syphilitic leptomeningitis about the pons would be much more probable, and this I believe was Dr. Pitt's hypothesis. Cancer, in the strict sense of the word, was obviously excluded in my case by the age of the patient and the general history and symptoms, apart from its not occurring as a symmetrical lesion. But some forms of sarcoma are more or less symmetrical in the skin, and, as I once saw, in both adrenal bodies; moreover the resistance of the nodes on my patient's skull to anti-syphilitic treatment raised the question of whether they might be tumours and not gummata; but they were not painful, did not involve the skin, were not vascular and increased much too slowly for sarcomata. The possibility of an exostosis in the

internal auditory canal compressing the nerves in their course through it was considered by Dr. Roberts, but I am not aware that any case of such a growth on both sides has been recorded. Lastly, the fact that the two cases published in 1862 were syphilitic confirms the same conclusion in my own patient's case, to which I was led by the fact of lues being present and by exclusion of other causes.

Double facial paralysis (*Diplegia facialis*) is a rare affection compared with the ordinary unilateral form of "Bell's palsy." Cases have been recorded by Sir Charles Bell himself, by Magendie, Christison, Gairdner, and by Mons. Davaine, who in 1852 published a series of collected cases in the 'Gazette médicale de Paris'[1] (p. 3, s. vii).

The monograph of Davaine contains eight cases collected from Romberg, Sir Charles Bell, Christison, and later authors of facial diplegia (paralysis of the portio dura on both sides), but in several the right or left palsy was successive, not simultaneous, and in none of them was deafness also present.

Nor do Dr. Wilks ('Diseases of the Nervous System,' 2nd ed., p. 567–8), Prof. Erb ('Ziemssen's Handbuch,' ii, s. 469), Dr. Bastian,[2] or the late Dr. Ross[3] refer to cases.

In Dr. Gowers' text-book on the same subject (vol. ii, p. 216) he remarks:—"In one case of this kind," viz. double facial paralysis, "in which there was also simultaneous paralysis of both auditory nerves (not aural), the onset occurred after exposure during sleep to a cold wind; but the patient had had syphilis."

In Dr. Bristowe's interesting volume of 'Clinical Lectures and Essays on Disease of the Nervous System' (p. 208) he narrates a case of diplegia facialis with deafness, but this was the result of fracture of the base of the skull.

Sir William Roberts quotes a case from the late Dr. Todd's 'Clinical Lectures on Nervous Diseases' (1854). It is num-

[1] This was reprinted as a pamphet of fifty-seven pages under the title 'De la Paralysie générale ou partielle des deux nerfs de la septième paire,' Paris, Thernot, 1852.

[2] 'Paralysis—Cerebral, Bulbar, and Spinal,' p. 452.

[3] 'Diseases of Nervous System,' vol. i, p. 477.

bered xvi, and occupies pp. 83—86; but the patient was not seen by Dr. Todd himself, he was under the care of Mr. Holthouse, whose report of the case is quoted. The paralysis of the right side occurred when the patient was twenty-nine years old, was accompanied by severe earache, and followed by otorrhœa and deafness. Subsequent deafness affected the left ear, and paralysis the left side of face. When seen (apparently only once) by Mr. Holthouse there was complete double facial paralysis and complete deafness, but only the right tympanum was destroyed, the left being apparently unaffected. Whether the inflammation of the middle ear on the right side was of syphilitic origin there is nothing to show.

This case, the one referred to by Dr. Gowers, the two above quoted from Sir William Roberts, and the present one are, therefore, the only ones I have found recorded of this remarkable condition.

Since, however, the above lines were written, Mr. Hutchinson has published a case of the same combination of double facial paralysis with absolute deafness in the 'Clinical Journal' for May 29th, 1895 (p. 78).

The patient was a widow woman (age not stated) who was living in the Cleveland Street Sick Asylum, and who had been frequently a patient of Dr. Hughlings Jackson. The paralysis of facial and auditory nerves came on nearly at the same time, and had lasted about three years. Here also, as in my case, there were swellings on the head, which "resembled rather solid œdema of the scalp than positive nodes," and the existence of lues was suspected. Mr. Hutchinson remarks that he has never before seen double paralysis of the portio dura with deafness, and says that most of the examples of (unilateral or bilateral) deafness with (unilateral) facial palsy from syphilis which he has seen have occurred within the second year after infection. He adds, "Probably there are no cases which more definitely demand prompt and efficient treatment than those in which deafness is threatened in the course of syphilis. Unless the hearing be restored within a week or ten days the patient will probably remain irremediably deaf."

LIST OF SPECIMENS ADDED TO THE PATHOLOGICAL MUSEUM

DURING THE YEAR 1894.

By L. E. SHAW, M.D., and E. C. PERRY, M.D.

1249 Laceration of the Liver.

A portion of the right lobe of a liver, showing several lacerations, the edges of which are covered with recent lymph, a similar material being deposited upon the capsule of the organ.

James H., æt. 53, was admitted under Mr. Howse, having been crushed against a wall by a load which fell from a passing waggon. Bronchitis and pleurisy supervened, and he died six days after the accident. At the autopsy blood was found in the peritoneal cavity, and there was an encysted collection of bile-stained serum, measuring about two pints, between the liver and diaphragm. *See Insp.*, 1890, No. 158.

1252 Liver wounded in the Operation for Empyema.

The lower part of a right lung thickly coated with lymph and adherent to the diaphragm, together with the adjacent portion of the right lobe of the liver. Just below the lung, at its hinder part, is seen a ragged opening in the diaphragm leading to a deep laceration in the liver. There is recent

blood-stained lymph on the surface of the liver around the margin of the laceration.

From James H., æt. 42, who suffered from an empyema following lobar pneumonia. An attempt was made to open the empyema by an incision in the ninth intercostal space. Three days later the patient died from purulent peritonitis.

1255 Fissure in the Liver.

A portion of a liver, showing upon its upper surface a shallow linear depression three inches in length running obliquely backwards and inwards from the notch marking the fundus of the gall-bladder. There are organised adhesions attaching the gall-bladder to the liver and duodenum.

Arthur B., æt. 48, was admitted under Dr. Pavy for gonorrhœal synovitis accompanied by persistent vomiting, from which three weeks later he died. He was not known to have received any abdominal injury. *See Insp.*, 1889, No. 363.

1259 A Deformed Liver.

A portion of the right lobe of a liver mounted to show its anterior edge, for a distance of three inches to the right of the gall-bladder, turned upwards and backwards, so as to be in close contact with the upper surface. Along the line of flexure and for some distance above it the capsule covering the upper surface of the organ is somewhat thickened.

Elizabeth B., æt. 44, was admitted under Dr. Perry with intestinal obstruction, for which colotomy was performed. She died seven weeks after the operation, and at the autopsy a carcinomatous stricture was found in the sigmoid flexure, and there were distension ulcers in the cæcum and ascending colon. *See Insp.*, 1891, No. 246.

1267 Localised Red Atrophy of the Liver.

A section of a liver, the greater portion of which is smooth, firm, and dark in colour, whilst at the upper part of its convex surface is a considerable area, and elsewhere are small scattered foci, in which the hepatic tissue has a light yellow colour, and is of soft consistency. Histologically the structure of these parts appears to be normal, the darker parts showing great dilatation of the capillaries, slight excess

of fibrous tissue, and almost complete atrophy of the hepatic cells. Under the microscope a few miliary tubercles are seen scattered through the substance of the organ.

Wellington S., æt. 41, was admitted under Dr. Pye-Smith for phthisis associated with laryngitis and ascites. He died four weeks after admission, and at the autopsy the liver was found to weigh 57 ounces, and there were miliary tubercles in the supra-renal capsules. The kidneys were hard and congested. *See Insp.*, 1894, No. 258.

1272 Fatty Liver in Phosphorus Poisoning.

A section from the right lobe of a liver, mounted to show the condition of extreme fatty degeneration. In the recent state the organ was pale in colour, and so light as almost to float in water.

Alice G., æt. 18, was admitted under Dr. Goodhart, having swallowed phosphorus paste, and died seven days after the accident. Whilst in the hospital she vomited frequently, had severe abdominal pain, and bled from the mouth and gums. The liver was felt to be enlarged. At the autopsy fatty degeneration was found in the muscles, and in the epithelium of the kidneys. *See Insp.*, 1891, No. 35.

1281 Nutmegged Liver.

A portion of the right lobe of a liver, partially injected, and showing its serous surface for the most part to be thickly covered with minute tags of fibrous tissue. On the anterior border and adjacent under surface the capsule of the organ is, as it were, sanded over with granules which are considerably smaller than miliary tubercles. Histologically the hepatic tissue presents the appearances produced by prolonged congestion of the intra-lobular veins, and the projections from the capsule are seen to consist of organised fibrin.

William R., æt. 24, was admitted under Dr. Taylor for cardiac dropsy of several months' duration, for the relief of which his legs were punctured. Cellulitis supervened, and the patient died four days after admission. At the autopsy the heart was found to weigh 24 ounces; the pericardium was universally adherent, and the aortic and mitral valves were fibrous and incompetent. The liver weighed 61 ounces, and was nutmegged. There were infarcts in the lungs, and the viscera generally were congested. *See Insp.*, 1894, No. 244.

1299 Multiple Abscesses in the Liver.

A portion of the right lobe of a liver showing upon its cut surface numerous excavations, which vary in size from a pin's head to a pea, and are partly filled with inspissated pus. Histologically the capsule of Glisson is found to contain an excess of small round cells, and in many places to present a minute abscess, in the centre of which is a mass of orange-coloured bile-pigment.

William B., æt. 39, was admitted under Dr. Goodhart with febrile jaundice of ten days' duration, having twice previously suffered from biliary colic. Vomiting and diarrhœa supervened, and the patient died ten days after admission. At the autopsy a calculus was found partially obstructing the common bile-duct, and the gall-bladder was distended, but neither ducts nor gall-bladder appeared to be ulcerated. *See Insp.*, 1888, No. 457; and *Guy's Hospital Gazette*, 1888.

1320 Gumma of the Liver.

A portion of the right lobe of a liver, the anterior half of which is occupied by a circumscribed yellowish mass of cartilaginous consistency. The capsule covering the gumma is thickened and firmly adherent to the adjacent diaphragm. Histologically the mass consists of fibrous tissue with areas of caseous and calcareous degeneration.

Francis F., æt. 59, was admitted under Dr. Taylor with intestinal obstruction, for the relief of which a right inguinal colotomy was performed. Three days later the patient died, and at the autopsy a malignant stricture was found in the colon just below its splenic flexure. The liver weighed fifty-two ounces, the left lobe being much hypertrophied. The testes were fibrous. *See Insp.*, 1892, No. 5.

1327 Syphilitic Fibrosis of the Liver.

A portion of a liver, the capsule of which is thickened and its shape deformed by irregular depressions and fissures producing bossy elevations upon its surface. The cut sections show the organ to be traversed by numerous white bands, which histologically are seen to consist of fibrous tissue invading the healthy liver-substance.

Charles N., æt. 23, was admitted under Dr. Goodhart with ascites of five months' duration, which had been relieved on many occasions by paracentesis abdominis. Two years previously he had had an attack of

jaundice, which lasted for five months. There were well-marked evidences of congenital syphilis. The patient died suddenly on the day following his admission, and at the autopsy the peritoneal cavity was found to contain thirteen pints of turbid yellow fluid. The spleen weighed 15½ ounces. *See Insp.*, 1890, No. 388.

1328 Syphilitic Scarring of the Liver.

A portion of the right lobe of a liver, the surface of which is marked by numerous irregular linear depressions, giving it a scarred and deformed appearance. The capsule is thin, and the cut section shows a small yellow gumma embedded in the substance of the organ.

At the bottom of some of the depressions in the liver, histological examination revealed the presence of contracting gummata.

1331 Caseous Nodules in the Liver.

A portion of the right lobe of a liver, showing two flattened nodules projecting slightly above the capsule. They measure about a quarter of an inch in diameter, and the section through one of them shows that it is embedded in the substance of the liver, from which it is separated by a well-defined margin. Histologically the nodules have the characters of caseating tubercle.

Henrietta P., æt. 11 months, was admitted under Mr. Lane in a moribund condition. She died the next day, and at the autopsy caseous masses were found in the lungs and bronchial glands. *See Insp.*, 1891, No. 92.

1334 Liver in Pernicious Anæmia.

A portion of a liver containing an excess of iron, as shown by the deep blue colour imparted to it by immersion in a solution of ferrocyanide of potassium acidulated by very dilute hydrochloric acid. The percentage of iron in the dried organ was estimated by Mr. Hopkins to be 1·038, an amount more than eleven times greater than the average normal percentage. Histological examination shows that the iron is principally distributed at the periphery of the lobule.

Richard F., æt. 55, was admitted under Dr. Hale White for well-marked pernicious anæmia of eight weeks' duration. The blood was found to contain 24 per cent. of the normal number of corpuscles, and 18 per

cent. of the normal quantity of hæmoglobin. The liver was slightly enlarged, and increased in size while under observation. Pericarditis supervened, and the patient died fourteen weeks after admission. At the autopsy the liver was found to weigh 66 ounces, and this organ as well as the kidneys and spleen contained a great excess of iron. *See Insp.*, 1893, No. 224; and *Guy's Hosp. Reps.*, vol. l, p. 349, Case 1.

1335 Aneurysms of the Hepatic Artery.

A portion of a liver with the structures in the portal fissure seen from the under surface of the organ. On the right branch of the hepatic artery, just at its origin, is seen an aneurysmal sac measuring three and a half by two and a half inches. The sac lies upon the upper part of the gall-bladder and compresses it, and on its external surface is seen the common bile-duct, the proximal portion of which is much dilated, as are also its branches throughout the liver. Upon the left branch of the hepatic artery is a similar and smaller aneurysm embedded in the substance of the liver. The aorta is healthy.

George L., æt. 18, was admitted under Dr. Hale White with lobar pneumonia affecting the right lung. Twenty days after admission the patient became jaundiced, and four days later pus was evacuated from the right pleural cavity. He died suddenly a month after admission, and at the autopsy the abdomen was found to contain several pints of blood-clot, the hæmorrhage having proceeded from a rent in the larger of the two aneurysms shown in the preparation. *See Insp.*, 1891, No. 117.

1344 Cylindrical-celled Carcinoma of the Liver.

A portion of a liver, showing a wedge-shaped deposit of firm white growth at the posterior part of the organ close to the inferior vena cava. Histologically the growth is a cylindrical-celled carcinoma.

Ellen G., æt. 36, was admitted under Dr. Taylor with an artificial anus formed eleven months previously for the relief of intestinal obstruction. An attempt was made to close the fistula, and the patient died from peritonitis. At the autopsy a malignant stricture was found in the descending colon, and there were several secondary deposits of growth in the liver. *See Insp.*, 1893, No. 229.

1345 Carcinoma of the Liver.

A portion of a liver mounted to show on its surface numerous irregular depressions, over which the capsule is

thickened and roughened by adhesions. Corresponding to these depressions are deposits of scirrhous growth, which is seen on the cut surface to infiltrate and in a great measure to replace the substance of the liver. Histologically the growth is a spheroidal-celled carcinoma with much stroma.

Maria O., æt. 50, was admitted under Dr. Goodhart with an enlarged liver and a contracting growth in her right breast, the nipple of which had been drawn in for nine years. She died eleven days after admission, and at the autopsy the lungs were found to be puckered by the contraction of secondary deposits. *See Insp.*, 1891, No. 18; and *Prep.* A 3351.

1346 Spheroidal-celled Carcinoma of the Liver.

A slice taken from the right lobe of a liver, the cut surface of which shows the organ to be extensively infiltrated by a white deposit, which histologically has the characters of a spheroidal-celled carcinoma with scanty stroma. The capsule is in parts thickened, and presents irregular depressions.

Elizabeth M., æt. 38, was admitted under Dr. Taylor with a painful and distended abdomen. She died two weeks after admission, and at the autopsy 50 ounces of fluid were found in the peritoneal cavity. The liver, which was uniformly infiltrated with growth, weighed 70 ounces. In the right breast there was a hard mass of carcinoma an inch and a half in diameter. There were no other secondary deposits. *See Insp.*, 1894, No. 10.

1356 Carcinoma of the Liver invading the Vena Cava.

A portion of a liver mounted to show a mass of growth invading and partially occluding the inferior vena cava. On the cut surface are seen numerous areas of white growth, which have the histological characters of cylindrical-celled carcinoma.

Charles M., æt. 67, was admitted under Dr. Taylor with ascites and œdema of the legs, from which he had suffered for four months. Paracentesis abdominis was performed, and the patient died ten days after his admission. At the autopsy malignant growth was found in the stomach, and there were secondary deposits in the lung, pleura, and bronchial glands. *See Insp.*, 1891, No. 124.

1357 Cirrhosis and Carcinoma of the Liver.

An antero-posterior slice from the right lobe of a liver, showing at the upper part its substance replaced by numerous closely aggregated nodules of growth embedded in a fibrous stroma. The rest of the hepatic tissue shows the lobules to be separated from each other by an excessive thickening of connective tissue. Histologically the growth is a spheroidal-celled carcinoma, and the liver is cirrhotic.

John R., æt. 49, was admitted under Dr. Goodhart for ascites of two months' duration. He had been a heavy drinker for fifteen or sixteen years, but had abstained from alcohol for six years before his death. Blood-stained serum was withdrawn from the peritoneal cavity and from the right pleura. The patient died three weeks after his admission, and at the autopsy secondary deposits were found in the bronchial and portal glands. It was thought that the liver was the primary seat of the growth. *See Insp.*, 1892, No. 410.

1358 Carcinoma and Cirrhosis of the Liver.

A portion of a liver, the cut surface of which shows a great excess of fibrous tissue replacing the hepatic substance. A main branch and many of the smaller branches of the portal vein are filled with thrombus, which under the microscope is seen to consist of large spheroidal cells embedded in a scanty stroma. The liver-substance is cirrhotic, and in the fibroid tissue are observed alveoli of various sizes filled with cells similar to those forming the thrombus.

Isaac B., æt. 68, was admitted under Dr. Pye-Smith with an enlarged liver, which was painful and nodular. Ascites supervened, and the patient died comatose sixteen days after admission. At the autopsy a healed ulcer was found upon the lesser curvature of the stomach. The liver weighed 71 ounces, and was the only organ in which malignant deposit was discovered. *See Insp.*, 1891, No. 273.

1362 Sarcoma of the Liver.

Portions of a liver mounted to show numerous soft white nodules, varying in size from a swan-shot to a cherry, some of which are deeply embedded in the organ, whilst others project beneath the capsule. Histologically the deposit consists of circumscribed nodules of small round cells. The liver is fatty.

Presented by Mr. Lawford Knaggs, 1888.

1373 Retention Cyst of the Liver.

A portion of the left lobe of a liver, incised to show immediately beneath the capsule a cyst which is about the size of a walnut, and in the recent state contained a colourless opalescent fluid. The wall of the cyst, which presents numerous lacunæ, is thin, and histologically is found to be composed of fibrous tissue having no epithelial covering.

John G., æt. 62, was admitted under Mr. Jacobson with a sarcoma of the femur, for which amputation was performed. The patient died a month after the operation, and at the autopsy secondary deposits were found in the ileum and lungs. The liver weighed 51 ounces, and appeared normal except for the cyst shown in the preparation. *See Insp.*, 1894, No. 170.

1374 Multiple Hydatids of the Liver.

The liver of a child, containing numerous hydatid cysts, some immediately beneath the capsule, others more deeply embedded in the organ. On section the cavities are seen to vary from an inch to a third of an inch in diameter, and to be lined with a delicate hydatid membrane.

From a child who died in the Evelina Hospital in 1888. The lungs were similarly affected.

Presented by Dr. GOODHART.

1382 Hydatid of the Liver.

A portion of the right lobe of a liver, showing at its posterior part a hydatid cyst measuring two inches in diameter, the walls of which are composed of fibro-calcareous material. The contents of the cyst are formed of a putty-like mass, embedded in which are seen pieces of hydatid membrane. Histological examination shows this mass to consist of granular débris, fat, and cholesterine.

Daniel H., æt. 61, was admitted under Mr. Davies-Colley with cerebral symptoms attributed to injury. He was trephined, and died comatose on the eighth day after the operation. At the autopsy the cerebral vessels were found to be very atheromatous, and there were numerous patches of softening in the brain. *See Insp.*, 1890, No. 232.

1389 Gall-bladder and Bile-ducts distended by Blood; Cholecystotomy.

A gall-bladder with the cystic, hepatic, and common bile-ducts, together with the adjacent portions of the liver and anterior abdominal wall. The gall-bladder has been laid open, and shows a longitudinal laceration about two and a half inches in length on its anterior wall towards the right side. The rent extends through the coats of the bladder, and its edges are covered by adherent blood-clot. The cystic and the lower part of the common bile-duct are slightly dilated, the rest of the latter and the hepatic ducts being enormously distended. The fundus of the gall-bladder has been sutured to the abdominal wall, and has been opened a little to the left of its long axis. The drainage-tube inserted at the operation remains *in situ*. Below are mounted the blood-clots removed at the autopsy from the gall-bladder and the hepatic ducts, the former measuring two and a half and the latter one and a quarter inches transversely.

1391 Deformity and Malposition of the Gall-bladder.

A portion of the right lobe of a liver mounted to show the gall-bladder dilated and turned to the left, so that its greater part forms an egg-shaped tumour three inches in length, lying parallel to and projecting beyond the anterior edge of the organ. Running upwards and to the right from the notch for the gall-bladder there is a deep furrow on the anterior surface of the liver.

John W., æt. 58, was admitted under Dr. Perry for persistent vomiting, and died from carcinoma of the stomach. At the autopsy a malignant stricture of the pylorus was discovered, and there were numerous filiform polypi in the ileum. *See Insp.*, 1894, No. 363.

1393 Distension of the Gall-bladder.

The anterior half of a liver, mounted to show the gall-bladder projecting as a pear-shaped sac four inches beyond the margin of the organ. The liver is small, and its capsule is wrinkled, and presents a slight furrow with local thickening thought to be due to the pressure of a belt.

John H., æt. 41, was admitted under Mr. Durham with a fracture of the neck of the left femur, from the effects of which he died five weeks later. At the autopsy the liver was found to weigh 39 ounces, and the bile-ducts were free from obstruction. *See Insp.*, 1891, No. 418.

1395 Distended and Inflamed Gall-bladder in Enterica.

A portion of a liver with the gall-bladder mounted to show the latter greatly distended, so as to measure in the recent state seven and a half inches in length. Its wall is thin, and its serous surface presents a deposit of granular lymph.

Henry A., æt. 19, was admitted under Dr. Hale White during a relapse of typhoid fever, which proved fatal at the end of the seventh week. He suffered from profuse diarrhœa, and, for the two days preceding his death, from frequent vomiting. At the autopsy the gall-bladder was found to contain 15 ounces of watery bile, which, upon slight pressure, flowed freely into the duodenum. *See Insp.*, 1890, No. 435.

1396 Suppurating Gall-bladder.

A portion of the right lobe of a liver, showing the gall-bladder to be replaced by a mass of inflammatory tissue to which the omentum was adherent. In the midst of this mass is a cavity from which a yellow rod has been passed along a sinus into the duodenum, which it enters about an inch from the pylorus. On the reverse of the specimen is seen a large hydatid cyst projecting from the under surface of the liver.

Matilda M., æt. 69, was admitted under Dr. Perry with the signs of acute peritonitis. She had suffered for the previous three months from occasional attacks of abdominal pain and vomiting. She died twelve hours after admission, and at the autopsy an abscess was found in the region of the gall-bladder communicating with the peritoneal cavity, which contained 30 ounces of yellowish purulent fluid. *See Insp.*, 1892, No. 272.

1399 Cholecysto-duodenal Fistula; Gall-stone impacted in the Ileum.

A thickened and contracted gall-bladder with the duodenum, to which it is firmly adherent. Between the two viscera there is a fistulous communication easily admitting the middle finger. The opening in the intestine is situated about an inch from the pyloric ring. Below is mounted a portion of ileum in which is firmly impacted a gall-stone

measuring an inch and a half in length by one inch in breadth.

Emma M., æt. 59, was admitted under Dr. Hale White for intestinal obstruction. She had suffered from obstipation, vomiting, and abdominal pain for six days. Two days after admission laparotomy was performed and an artificial anus established in the small intestine. The patient died six hours later, and at the autopsy the stone was found to be impacted 33 inches above the ileo-cæcal valve. *See Insp.*, 1895, No. 24.

1406 Cylindrical-celled Carcinoma of the Gall-bladder.

A portion of the anterior edge of a liver, showing in the situation of the gall-bladder a large mass of malignant growth, with which the omentum is firmly united. At the junction of the mass with the edge of the liver there is an opening made by operation into the cavity of the gall-bladder, which contains several calculi. On the reverse of the specimen is seen the upper part of the duodenum, the mucous membrane of which is stretched over the growth and ulcerated for a distance of three inches, beginning half an inch from the pylorus. The cut surface of the liver shows several secondary deposits, which histologically have the characters of cylindrical-celled carcinoma, some of the cells of which have undergone slight colloid change.

Fanny P., æt. 37, was admitted under Mr. Lane with an abdominal tumour in the right hypochondriac region, having previously suffered from occasional attacks of jaundice. Laparotomy was performed, and secondary deposits were observed in the liver. The gall-bladder, which was distended with bile, was incised, but no calculi were discovered. The patient died five weeks after the operation. Secondary deposits of growth were found in the peritoneum. *See Insp.*, 1893, No. 411.

1408 Carcinoma of the Gall-bladder.

A portion of the right lobe of a liver with the gall-bladder mounted to show the latter surrounded by a mass of firm growth, to which is adherent a portion of the omentum and of the transverse colon. The primary mass of growth has invaded the adjacent substance of the liver, and there are secondary nodules scattered throughout the organ. Histologically the growth is a spheroidal-celled carcinoma.

Sarah C., æt. 65, was admitted under Dr. Perry with jaundice and a tumour in the right hypochondrium, having suffered from severe abdominal pain for the three months preceding her admission. She died three weeks later; and at the autopsy the gall-bladder was found to contain several gall-stones, and there were secondary deposits of growth in the peritoneum. *See Insp.*, 1892, No. 303.

1409 Carcinoma of the Gall-bladder.

A portion of a liver with the gall-bladder, from the mucous membrane of which project several rounded masses of growth, the largest being about three quarters of an inch in diameter. Around the neck of the gall-bladder are several glands enlarged and infiltrated by malignant deposit, which histologically has the characters of a spheroidal-celled carcinoma. The liver is granular upon the surface, and under the microscopic shows marked cirrhosis.

George J., æt. 65, was admitted under Dr. Taylor with ascites and general dropsy, and died fourteen days after admission. At the autopsy the arteries were found to be extremely atheromatous, and the heart was hypertrophied and dilated. There was a carcinomatous ulcer in the descending colon, and there were numerous secondary deposits in the peritoneum and liver. *See Insp.*, 1891, No. 178.

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,

&c. &c.,

IN THE YEAR 1894.

University of Oxford.

Final Examination for the Degrees of Bachelor of Medicine and Surgery.

H. A. Munro.

University of Cambridge.

Final Examination for the Medical and Surgical Degrees.

Part I.

C. S. Bond.	C. E. Cooper.	T. P. King.
L. C. Burrell.	E. E. Henderson.	E. A. Peters.
A. V. Clarke.		

Part II.

C. S. Bond.	G. H. Field.	B. W. M. A. Key.
A. V. Clarke.	E. E. Henderson.	T. P. King.
A. J. Collis.	C. L. Hopkins.	E. A. Peters.
F. N. Day.		

Second Examination for the Medical and Surgical Degrees.

Part I.

W. J. Lindsay. | F. G. Thomas.

Part II.

A. B. Carter. | C. Jephcott.

The Degree of Doctor of Medicine was conferred upon—

D. W. Samways. | C. R. A. Sutton.

Examination in Sanitary Science.

V. J. Hodgson. | E. P. Manby.

University of London.

Examination for the Degree of Doctor of Medicine.

S. G. Floyd.	G. L. Kemp.	A. J. Sharp.
J. A. Howard.	T. D. Lister.	W. C. Swayne.
J. W. F. Jewell.	A. E. Norburn.	T. R. Taylor.
W. J. Johnson.	C. S. Pantin.	J. Young.

Examination for the Degree of Doctor in State Medicine.

B. W. Hogarth.

Examination for the Degree of Master in Surgery.

H. W. Webber.

Examination for the Degree of Bachelor of Surgery.

First Division.

Hugh Davies.

Obtained the Gold Medal.

C. E. Carpmael.	G. S. Hovenden.	E. C. Taylor.
J. St. T. Clarke.	E. Huntley.	

Second Division.

E. L. Adams.	H. W. Collier.	T. D. Manning.
A. P. Allan.	A. C. Hovenden.	F. J. Steward.

Final Examination for the Degree of Bachelor of Medicine.

May.

Second Division.

E. L. Adams.	E. Huntley.	A. Miller.
A. P. Allan.	G. L. Kemp.	L. A. Parry.

October.

First Division.

A. H. Leete.

Obtained the Scholarship and Gold Medal in Medicine.

F. J. Steward.

Obtained First-Class Honours in Medicine, Obstetric Medicine, and in Forensic Medicine.

G. E. Manning.

Obtained First-Class Honours in Medicine and in Forensic Medicine.

E. C. Taylor.

Obtained First-Class Honours in Obstetric Medicine and Honours in Medicine.

Second Division.

F. G. Hopkins.

Obtained Honours in Medicine and in Forensic Medicine.

Hugh Davies.

Obtained Honours in Medicine.

A. C. Hovenden.

Obtained Honours in Medicine.

A. Armer. | H. W. Collier. | T. D. Manning.
C. E. Carpmael. | G. S. Hovenden.

Intermediate Examination in Medicine.

January.

Second Division.

J. Robertson. | E. T. Scowby. | H. M. Wise.

Excluding Physiology.

Second Division.

A. H. Spicer. | W. L. Stuart.

Physiology only.

Second Division.

J. R. Steinhaeuser.

July.

Honours Examination.

V. E. Collins.

Obtained the Gold Medal in Physiology.

G. K. Levick.

Obtained Honours in Anatomy, and in Materia Medica and Pharmaceutical Chemistry.

J. Howell.

Obtained Honours in Materia Medica and Pharmaceutical Chemistry.

Pass List.

First Division.

F. W. Goldie.

Second Division.

A. Earnshaw. | H. Leader. | E. Ivens Spriggs.
E. Fisk. | J. T. Roberts. | S. Whicher.
R. Kay.

Physiology only.

Second Division.

R. H. Ashwin. | A. H. Spicer.

Preliminary Scientific (M.B.) Examination.

July.

Entire Examination.

First Division.

J. Atkins. | J. T. Dunston. | W. B. Secretan.
F. W. Brook.

Second Division.

A. Fraser.	G. N. Meachen.	W. G. Stewart.
J. T. M. McDougall.	D. J. Munro.	F. D. Turner.

Chemistry and Physics only.

E. G. Andrew.	S. J. Ormond.	R. Spearman.
A. Moon.		

Biology only.

H. B. Foster.	W. W. Harrison.	T. Morgan.
D. S. Graves.		

Entire Examination.

First Division.

W. R. Cazenove.

Second Division.

A. E. Clarke.	H. Durbridge.	E. E. Parrett.

Chemistry and Physics only.

H. E. C. Fox. | W. W. Harrison.

Biology only.

R. M. Barron.	J. E. Dupigny.	E. T. Jensen.

Final Examination for the Degree of Bachelor of Science.

G. E. Richmond. | P. Turner.

University of Durham.

Final Examination for the Degrees of Bachelor of Medicine and Surgery.

C. H. Bryant.	W. F. Miller.	F. Zumbado.

Examination for the Degree of Doctor of Medicine for Practitioners of Fifteen Years' Standing.

A. Kinsey-Morgan.

Second Examination for the Degree of Bachelor of Medicine.

A. M. Rygate.

Obtained Honours.

Pass List.

J. J. Foster.

University of Edinburgh.

Examination for the Degree of Bachelor of Science (Department of Public Health).

J. M. Hobson.

Naval Medical Service.

G. T. S. Sichel.
Head of List.

Royal College of Physicians.

Admitted to the Fellowship.

H. Davy. | E. C. Perry. | J. W. Washbourn.

Final Examination for the License.

January.

T. Burrow.	G. S. Hovenden.	E. Reid.
J. G. B. Coleman.	G. Lawrence.	F. H. Simpson.
H. A. Duffett.	A. H. Leete.	E. C. Taylor.
R. H. Fagge.	W. C. C. Park.	E. S. Tuck.
E. S. Hoare.	F. G. M. Philps.	

April.

E. G. Annis.	G. H. Field.	L. A. Parry.
A. H. Card.	E. F. H. Hardenberg.	R. E. G. Phillips.
A. J. Collis.	A. Miller.	E. E. W. Roe.
A. G. Cooley.	W. F. Miller.	W. K. Steele.
F. Cowan.	T. I. Mills.	P. Wilmot.

July.

H. W. Beach.	C. H. Harding.	H. A. Munro.
C. E. Carpmael.	G. S. O. Howes.	J. R. Steinhaeuser.
A. B. Creak.	G. Jimenez.	W. E. Sturges-Jones.
E. S. Dukes.	C. R. Lucas.	H. J. L. Wales.
R. D. Evans.		

October.

A. Allport.	S. Copley.	R. H. Moon.
A. Armer.	E. H. Drake.	E. G. E. O'Leary.
V. H. Barr.	G. L. Eastes.	H. D. Packer.
C. S. Bond.	F. A. Evison.	E. A. Peters.
F. S. Brereton.	F. G. Hopkins.	B. G. Reynolds.
W. J. Burroughs.	A. K. Matthews.	W. E. Robinson.
C. R. Colley.		

Royal College of Surgeons.

Final Examination for the Fellowship.

D. M. Beddoe.	G. C. Hall.	T. G. Ouston.
F. P. S. Cresswell.	J. B. Leathes.	C. S. Pantin.
H. E. Counsell.	T. D. Lister.	G. T. S. Sichel.
H. E. Durham.	R. H. Luce.	

First Examination for the Fellowship.

H. T. S. Bell.	W. J. Hancock.	W. E. Plummer.
A. G. Butler.	J. Howell.	A. Shillitoe.
V. E. Collins.	A. H. Leete.	T. G. Stevens.
C. H. Fagge.	A. W. Ormond.	

Final Examination for the Membership.

January.

T. Burrow.
J. G. B. Coleman.
H. A. Duffett.
R. H. Fagge.
E. S. Hoare.
G. S. Hovenden.
G. Lawrence.
A. H. Leete.
W. C. C. Park.
F. G. M. Philps.
E. Reid.
F. H. Simpson.
E. C. Taylor.
E. S. Tuck.

April.

E. G. Annis.
A. H. Card.
A. J. Collis.
A. G. Cooley.
F. Cowan.
G. H. Field.
E. F. H. Hardenberg.
A. Miller.
W. F. Miller.
T. I. Mills.
L. A. Parry.
R. E. G. Phillips.
E. E. W. Roe.
W. K. Steele.
P. Wilmot.

July.

H. W. Beach.
C. E. Carpmael.
A. B. Creak.
E. S. Dukes.
R. D. Evans.
C. H. Harding.
G. S. O. Howes.
G. Jimenez.
C. R. Lucas.
H. A. Munro.
J. R. Steinhaeuser.
W. E. Sturges-Jones.
H. J. L. Wales.

October.

A. Allport.
A. Armer.
V. H. Barr.
C. S. Bond.
F. S. Brereton.
W. J. Burroughs.
C. R. Colley.
S. Copley.
E. H. Drake.
G. L. Eastes.
F. A. Evison.
F. G. Hopkins.
A. K. Matthews.
R. H. Moon.
E. G. E. O'Leary.
H. D. Packer.
E. A. Peters.
B. G. Reynolds.
W. E. Robinson.

Final Examination for the License in Dental Surgery.

A. L. Allworth.
U. E. Cave.
F. C. Constant.
T. S. Davison.
T. Godfrey.
P. F. Henry.
W. E. Hills.
H. G. Howitt.
A. H. H. Huckle.
E. Hutson.
G. W. Jones.
F. E. L. Lambert.
L. F. Morris.
L. G. Musgrave.
J. W. Pare.
J. Pilcher.
W. H. Pilcher.
E. W. Stabb-Johnson.
R. Thornton.
A. J. Tyrrell.
G. N. Willis.

MEDALLISTS AND PRIZEMEN.

JULY, 1895.

The Treasurer's Gold Medal for Clinical Medicine.
Francis James Steward, Old Charlton.

The Treasurer's Gold Medal for Clinical Surgery.
Francis James Steward, Old Charlton.

Gurney Hoare Prize for Clinical Study.
Arthur Henry Leete, Wellingborough.

Beaney Prize for Pathology.
Francis James Steward, Old Charlton.

Sands Cox Scholarship for Physiology.

George Ernest Richmond, Leicester, Prize £15.
Harold Wilson Bruce, Bow, *Prox. Accessit.*
Philip Turner, Earl's Court Gardens, Certificate.

Michael Harris Prize for Anatomy.

Frederick Edward Walker, Hampstead, Prize £10.
Harold Wilson Bruce, Bow, *Prox. Accessit.*

The Arthur Durham Prizes for Dissection.

First Year's Students.

John Tyley Montgomery McDougall, Saxmundham, Prize £5.
Russell Henry Jocelyn Swan, Camberwell, Certificate.
John Dell Bridger, Cricklewood, Certificate.
Walter Bernard Secretan, Croydon, Certificate.

Second Year's Students.

Frederick Edward Walker, Hampstead, Prize £15.
Vincent James Crawford, Blackheath, Certificate.
Lionel Edwin Charles Handson, Hampstead, Certificate.
Laurie Hugh McGavin, London, Certificate.

The Hilton Prize for Dissection.

John Ownsworth Garland, Barnsley.

Third Year's Prizes.

Robert Pugh Rowlands, Towyn, North Wales, Prize £25.
Robert Howard, Buckhurst Hill, Second Prize, £10.
Edmund Ivens Spriggs, Market Harborough, Certificate.
Charles Thompson Anderson, Cape Colony, Certificate.
John Thomas Roberts, Llanfachreth, Certificate.

Second Year's Prizes (1894).

Edmund Ivens Spriggs, Market Harborough, } equal,
Robert Pugh Rowlands, Towyn, } £17 10*s.* each.

Dental Students (1895).

Joseph Lewin Payne, Kennington Road, Prize £15.
Edward Claude Dimock, Waterbeach, Certificate.
Harold Dewe Mathews, Cheltenham, Certificate.

First Year's Prizes (1894).

Philip Turner, Earl's Court Gardens, First Prize, £50.
George Ernest Richmond, Leicester, Second Prize, £25.
Harold Wilson Bruce, Bow, Certificate.
William Taylor Milton, Blackheath, Certificate.

Dental Students (1895).

Kenneth Weldon Goadby, Reading, } equal, £5 each.
Norris Snell, Forest Hill, }
Arthur George Grant Plumley, Bristol, Certificate.
Edwin Ernest Darley Heesom, Redhill, Certificate.
Guy Chatterton, Peckham Rye, Certificate.

Practical Dentistry Prize.

Edward Claude Dimock, Waterbeach, Prize £10.

Open Scholarships in Arts.

Arthur Pearson, Brixton, £100.
George William Hare, Hastings, £50.

Dental Students.

Guy Chatterton, Peckham, £30.

Open Scholarships in Science.

Thomas Francis Rutter, Balham, £150.
John Thomas Dunston, Farnham, £60.
Francis William Brook, Peckham, Certificate.

Physical Society.

Session 1895–6.

Honorary President.—SAMUEL WILKS, M.D., F.R.S.

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Honorary Secretaries.—H. J. CAMPBELL, M.D., J. H. TARGETT, M.S.

PRIZEMEN FOR THE SESSION 1894–5.

The Society's Prize of £10 was given to Mr. E. Ivens Spriggs for his Paper upon "Food;" a Prize of £4 was also given to Mr. W. C. C. Pakes for his Paper upon "Diphtheria."

An Extra Prize of £5, given by the Treasurer, was awarded to Mr. B. A. Richmond, M.B., B.Sc., for his Essay upon "The Pathology of Dropsy."

CLINICAL APPOINTMENTS HELD DURING THE YEAR 1894.

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W. J. Harris.	G. F. Still.	J. A. Howard.
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H. M. Stewart.	H. A. Duffett.	

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O. Beven.
J. Howell.
E. J. O'Meara.
A. M. Rygate.
J. N. Gardiner.
H. R. Marsh.
N. Y. Lower.
C. J. Francis.

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F. C. Hitchins.
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F. Zumbado.
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W. Tyson.
C. C. Stead.

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C. E. Carpmael.
R. H. Luce.
C. S. Bond.
E. A. Peters.
F. S. Brereton.
B. G. Reynolds.
J. R. Steinhaeuser.
H. W. Collier.
W. E. Robinson.
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L. C. Burrell.
C. J. Kearney.
G. S. Hovenden.
E. C. Taylor.

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A. H. Leete.
W. Tyson.
R. O. Moon.
S. G. Graham.
T. M. Nicholson.
C. B. Statham.
R. Henderson.

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P. N. Vellacott.
C. H. Bryant.
E. Coleman.
G. Gocher.
W. E. Plummer.
A. S. J. Pearse.
W. Tyson.
W. J. Rowland.
A. W. Ormond.
C. A. C. Askew.

CLERKS TO THE ANÆSTHETISTS.

A. H. Card.
E. S. Tuck.
A. G. Cooley.
H. Hewetson.
A. Miller.
A. Allport.
R. Henderson.
N. Instone.
A. B. Creak.
W. E. Robinson.
E. S. Hoare.
G. S. Hovenden.
H. J. L. Wales.
C. M. Beadnell.
R. D. Evans.
G. L. Eastes.
J. C. S. Peatson.
H. Davies.
J. J. Coleman.
A. Reeves.
H. W. Collier.
T. H. B. Yorath.
H. A. Fotherby.
A. V. Clarke.
A. K. Matthews.
B. A. Richmond.
H. J. Spon.
M. A. Key.
A. P. Beddard.
T. M. Thomas.
W. J. Rowland.
D. M. Beddoe.
E. F. H. Hardenberg.
F. Loud.
E. A. Peters.
H. W. Beach.
E. C. Taylor.
C. E. Cooper.
A. P. Rainbird.
J. C. V. Wilkins.
A. L. Allworth.
L. Slater.
J. R. Steinhaeuser.
G. Jimenez.
E. E. Henderson.
T. P. King.
E. R. Fothergill.
L. C. Burrell.
A. J. Mathison.
S. Copley.
C. R. Lucas.
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G. Gocher.
F. J. Steward.
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E. Coleman.
M. P. Jones.
C. C. Jenkins.
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E. E. W. Roe.
C. J. Kearney.
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P. R. Lowe.
F. Zumbado.
C. A. Ensor.
R. F. Clark.
R. H. Moon.
P. E. Tresidder.
S. Infield.
F. A. Evison.
J. G. O. Lane.
R. O. Moon.

DENTAL SCHOOL.

APPOINTMENTS HELD DURING THE YEAR 1894.

DENTAL HOUSE SURGEONS.

J. B. Parfitt.
S. Keele.
G. B. Webb.
F. C. Constant.
W. R. Wood.
P. F. Henry.

ASSISTANT DENTAL HOUSE SURGEONS.

P. F. Henry.
U. E. Cave.
F. E. L. Lambert.
T. R. Chambers.
F. C. Constant.
E. Morgan.

DEMONSTATORS IN THE CONSERVATION ROOM.

G. H. Bowden.
E. Morgan.
A. J. Collett.
D. S. Stevens.
E. W. Stabb Johnson.
T. S. Davison.
T. R. Chambers.
W. E. Hills.
A. Cahill.
U. E. Cave.
L. F. Morris.
W. H. Trick.
C. A. H. Keall.

CURATORS OF THE LABORATORY.

D. S. Stevens.
F. C. Beaumont.
J. H. Walters.
E. C. Dimock.
C. A. H. Keall.

ASSISTANT DEMONSTRATOR OF DENTAL MICROSCOPY.

H. Wallis.

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E. Ashby.
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J. W. Goddard.
J. A. Y. Donston.
J. L. Payne.
H. D. Mathews.
C. A. H. Keall.
E. A. Longhurst.
F. E. Händel.
J. E. Dupigny.
E. W. Smith.
H. C. Cowles.
A. G. G. Plumley.
T. D. E. Goodman.
D. G. Worts.
H. R. Pratt.
D. S. Stevens.
H. King.
J. W. Lewis.
B. E. James.
A. Read.
T. E. Norton.
C. D. Outred.
F. C. Beaumont.
A. De Mierre.
E. E. Heesom.
G. S. Simpson.
B. F. Henry.
W. Briant.
L. TaBois.
G. Chatterton.
H. Charnock.
E. R. Tebbitt.
N. Denham.
N. H. Oliver.
B. B. Westlake.
A. F. A. Howe.
A. W. Penrose.
H. S. Crapper.
B. G. Tasker.
W. J. M. Lacey.
E. G. W. Wallis.
E. Coltman.
E. W. West.
G. G. Ellis.
H. Knight.

DRESSERS IN THE GAS ROOM.

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A. L. Allworth.
W. E. Hills.
W. H. Trick.
A. Cahill.
L. F. Morris.
A. F. A. Howe.
E. R. Tebbitt.
H. King.
J. A. Y. Donston.
J. W. Lewis.
F. Mühlenkamp.
U. E. Cave.
T. Godfrey.
J. W. Pare.
A. B. Ryle.
H. Wallis.
A. J. Collett.
A. E. B. Crosby.
E. Morgan.
J. W. Goddard.
C. J. Hinchliff.
N. Denham.
H. P. Taylor.
C. H. Charter.
E. Picnot.
E. W. Stabb Johnson.
O. Armer.
S. Harrison.
C. A. H. Keall.
A. Cahill.
H. D. Mathews.
A. W. Penrose.
F. E. Handel.
E. E. D. Heesom.
D. S. Stevens.

DRESSERS IN THE CONSERVATION ROOM.

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C. J. Hinchliff.
A. H. H. Huckle.
G. N. Willis.
O. Armer.
A. Read.
F. C. Beaumont.
A. T. Tyrrell.
W. H. Trick.
F. Mühlenkamp.
H. S. Crapper.
C. A. H. Keall.
H. Wallis.
R. H. Stevens.
E. R. Tebbitt.
R. G. Edey.
D. G. Worts.
E. E. D. Heesom.
E. A. Longhurst.
G. S. Simpson.
E. G. W. Wallis.
F. E. Händel.
E. W. West.
H. C. Cowles.
B. G. Tasker.
L. Ta Bois.
A. De Mierre.
S. W. Cock.
G. Chatterton.
C. Lee.
H. R. Pratt.
G. Naish.
S. J. Evans.

J. W. Pare.
A. J. Collett.
H. Howitt.
F. O. Stoker.
N. Denham.
H. D Mathews.
N. H. Oliver.
E. W. Stabb Johnson.
J. E. O. Brawn.
G. W. Jones.
H. P. Taylor.
D. S. Stevens.
A. F. A. Howe.
J. W. Lewis.
A. W. Penrose.
E. Hutson.
A. L. Mercado.
W. Briant.
W. J. M. Lacey.
E. Ashby.
J. W. Goddard.
H. King.
E. D. Bascombe.
A. E. B. Crosby.
A. G. G. Plumley.
A. W. Penrose.
J. M. C. Jacobs.
G. D. Slater.
A. E. Oddy.
L. S. Hounsell.
K. W. Goadby.
E. C. Brown.
B. F. Henry.

J. A. Y. Donston.
E. Picnot.
H. Cook.
A. L. Allworth.
B. B. Westlake.
C. D. Outred.
A. B. Ryle.
R. Thornton.
C. H. Charter.
W. H. Pilcher.
E. Ashby.
J. H. Walters.
E. C. Dimock.
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T. D. E. Goodman.
N. Snell.
L. Bidlake.
E. W. Smith.
J. E. Dupigny.
C. S. Brookhouse.
E. Coltman.
C. J. Hinchliff.
S. Harrison.
E. B. Dowsett.
B. F. Henry.
H. Knight.
G. G. Ellis.
C. N. Doyle.
W. H. Loosely.
A. Styer.
C. H. Knowles.

GUY'S HOSPITAL.

MEDICAL AND SURGICAL STAFF.

1895.

Consulting Physicians.
S. Wilks, M.D., LL.D., F.R.S.
F. W. Pavy, M.D., LL.D., F.R.S.

Consulting Obstetric Physicians.
H. Oldham, M.D.
J. Braxton Hicks, M.D., F.R.S.

Physicians.
P. H. Pye-Smith, M.D., F.R.S.
Frederick Taylor, M.D.
J. F. Goodhart, M.D.
W. Hale White, M.D.

Assistant Physicians.
G. Newton Pitt, M.D.
E. C. Perry, M.D.
L. E. Shaw, M.D.
J. W. Washbourn, M.D.

Obstetric Physician.
A. L. Galabin, M.D.

Assistant Obstetric Physician.
P. Horrocks, M.D.

Aural Surgeon.
W. Laidlaw Purves, Esq.

Dental Surgeons.
F. Newland-Pedley, Esq.
W. A. Maggs, Esq.
J. H. Badcock, Esq.

Medical Registrar.
J. H. Bryant, M.D.

Consulting Surgeons.
J. Birkett, Esq.
Thomas Bryant, M.Ch.

Consulting Ophthalmic Surgeon.
Charles Bader, Esq.

Surgeons.
H. G. Howse, M.S.
N. Davies-Colley, M.C.
R. Clement Lucas, B.S.
C. H. Golding-Bird, M.B.

Assistant Surgeons.
W. H. A. Jacobson, M.Ch.
Charters J. Symonds, M.S.
W. Arbuthnot Lane, M.S.
L. A. Dunn, M.S.

Ophthalmic Surgeon.
C. Higgens, Esq.

Assistant Ophthalmic Surgeon.
W. A. Brailey, Esq.

Instructor in Anæsthetics.
Tom Bird, Esq.

Anæsthetists.
J. F. W. Silk, M.D.
E. T. E. Hamilton, M.D.
H. F. Lancaster, M.D.
C. J. Ogle, Esq.
G. Rowell, Esq.

Surgical Registrar.
G. B. Smith, B.S.

Warden of the College.
Mr. Dunn.

Lying-in Charity.
Dr. Galabin and Dr. Horrocks.

Dean of the Medical School, and Curator of the Museum.
Dr. Shaw.

LECTURERS AND DEMONSTRATORS.

Clinical Medicine	The Physicians and Assistant Physicians.
Clinical Surgery	The Surgeons and Assistant Surgeons.
Medicine	Dr. Pye-Smith and Dr. Taylor.
Practical Medicine	Dr. Bryant.
Surgery	Mr. Howse and Mr. Davies-Colley.
Practical Surgery	Mr. Targett.
Operative Surgery	Mr. Dunn.
Midwifery and Diseases of Women	Dr. Galabin.
Practical Obstetrics	Dr. Horrocks.
Mental Diseases	Dr. Savage.
Ophthalmic Surgery	Mr. Higgens and Mr. Brailey.
Dental Surgery	Mr. Newland-Pedley.
Aural Surgery	Mr. Laidlaw Purves.
Diseases of the Skin	Dr. Perry.
Diseases of the Throat	Mr. Symonds.
Electro-Therapeutics	Dr. Pitt.
Anæsthetics	Mr. Tom Bird and Dr. Silk.
Hygiene and Public Health	Mr. Turner, Dr. Washbourn, and Mr. Hopkins.
Pathology	Dr. Pitt.
Morbid Anatomy	Dr. Pitt, Dr. Perry, and Dr. Shaw.
Morbid Histology	Dr. Pitt and Mr. Targett.
Bacteriology	Dr. Washbourn.
Forensic Medicine	Dr. Stevenson.
Anatomy	Mr. Lucas and Mr. Jacobson.
Practical Anatomy	Mr. Fripp, Dr. Hamilton, and Mr. Steward.
Physiology	Dr. Washbourn and Dr. Starling.
Practical Physiology	Dr. Starling, Dr. Fawcett, and Mr. Hopkins.
Materia Medica and Therapeutics	Dr. Hale White.
Practical Pharmacy	The Hospital Dispenser.
Chemistry	Dr. Stevenson and Mr. Groves.
Practical Chemistry	Mr. Groves, Mr. Wade, and Mr. Hopkins.
Experimental Physics	Professor Reinold, F.R.S., and Mr. Wade.
Biology	Mr. Beddard, F.R.S., Dr. Campbell, and Mr. Steward.
Psychology	Dr. Savage and Dr. Rivers.

The Hospital contains 695 beds, of which 500 are in constant occupation. Special Classes are held for Students preparing for the Examinations of the Universities and other Higher Examinations.

APPOINTMENTS.

All Hospital Appointments are made strictly in accordance with the merits of the Candidates, and without extra payment.

OPEN SCHOLARSHIPS IN ARTS AND SCIENCE.

Four Open Scholarships, two in Arts and two in Science, will be offered for competition in September, 1895.

Entrance Scholarships in Arts.

1. A Scholarship of £100, open to Candidates under 20 years of age.
2. A Scholarship of £50, open to Candidates under 25 years of age.

Entrance Scholarships in Science.

1. A Scholarship of £150, open to Candidates under 25 years of age.
2. A Scholarship of £60, open to Candidates under 25 years of age.

PRIZES

Are awarded to Students in their various years, amounting in the aggregate to more than £450.

DENTAL SCHOOL.

The Dental School is attached to the Hospital, which affords to Students all the instruction required for a Licence in Dental Surgery.

COLLEGE.

The Residential College accommodates about 50 Students in addition to the Resident Staff of the Hospital, and contains a large Dining Hall, Reading Rooms, and Gymnasium for the use of the Students' Club.

For Prospectus and further information apply to the Dean, Dr. Shaw, Guy's Hospital, London, S.E.

GUY'S HOSPITAL.

THE STAFF OF THE DENTAL SCHOOL.

1895.

Dental Surgeons.

F. Newland-Pedley, F.R.C.S., L.D.S.E.
W. A. Maggs, L.R.C.P., M.R.C.S., L.D.S.E.
J. H. Badcock, L.R.C.P., M.R.C.S., L.D.S.E.

Assistant Dental Surgeons.

R. Wynne Rouw, L.R.C.P., M.R.C.S., L.D.S.E.
H. L. Pillin, L.D.S.E.
G. O. Richards, M.R.C.S., L.D.S.E.
A. E. Baker, L.R.C.P., M.R.C.S., L.D.S.E.
M. F. Hopson, L.D.S.E.
H. Stoner, L.R.C.P., M.R.C.S., L.D.S.E.

Anæsthetists.

J. F. W. Silk, M.D.
F. W. Cock, M.D., M.S.
H. F. Lancaster, M.D.
W. J. Scott, M.B., B.S.
C. J. Ogle, M.R.C.S.
E. T. E. Hamilton, M.D.

Lecturers.

Dental Surgery and Pathology.—Mr. Newland-Pedley.
Dental Anatomy and Physiology.—Mr. Maggs.
Dental Mechanics.—Mr. Wynne Rouw.
Metallurgy.—C. E. Groves, F.R.S.
Operative Dental Surgery.—Mr. Badcock.

Demonstrators.

Dental Microscopy.—Mr. Baker.
Dental Mechanics.—Mr. Pillin.

Dean.—Dr. Shaw.

ASTLEY COOPER PRIZE.

The next Triennial Prize of Three Hundred Pounds,

Under the Will of the late SIR ASTLEY P. COOPER, Bart.,

WILL BE AWARDED TO

THE AUTHOR OF THE BEST ESSAY OR TREATISE ON

"*The Diseases of Joints caused by Syphilis and Gonorrhœa.*"

THE Conditions annexed by the Testator are, "That the Essays or "Treatises to be written for such Prize shall contain original experi- "ments and observations, which shall not have been previously pub- "lished, *and that each Essay or Treatise shall* (as far as the subject "shall admit of) *be illustrated by preparations and drawings*, which "preparations and drawings shall be added to the Museum of Guy's "Hospital, and shall, together with the Work itself and the sole and "exclusive interest therein and the copyright thereof, become hence- "forth the property of that Institution, and shall be relinquished and "transferred as such by the successful candidate."

And it is expressly declared in the Will "That no Physician or "Surgeon, or other officer for the time being, of Guy's Hospital or of "St. Thomas's Hospital, in the Borough of Southwark, nor any person "related by blood or affinity to any such Physician or Surgeon, for "the time being, or to any other Officer for the time being in either "of the said Hospitals, shall at any time receive or be entitled to "claim the Prize." But, with the exceptions here referred to, this Prize is open for competition to the whole world.*

Candidates are informed that their Essays, either legibly written or type-written, in the English Language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy's Hospital on or before January 1st, 1898, addressed to the Physicians and Surgeons of Guy's Hospital.

Each Essay or Treatise must be distinguished by a Motto, and accompanied by a sealed envelope containing the name and address of the Writer. None of the envelopes will be opened except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations or drawings, will remain at the Museum of Guy's Hospital until claimed by the respective writers or their agents.

* The Prize cannot be awarded to any Essay which is the joint production of two or more authors.

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